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CONTENTS

		PAGE
Experimental studies on vitreous detachment	*L. von Sallmann*	1349
Gliomas of the retina	*E. M. Parkhill and W. L. Benedict*	1354
Choked discs in types of blood dyscrasia	*C. H. Watkins, H. P. Wagener, and R. W. Brown*	1374
Experimental corneal transplantation	*M. Wiener and H. D. Rosenbaum*	1384
Toxic syndrome of ocular tuberculosis	*C. Charlin*	1392
Value of gonioscopy in glaucoma	*M. U. Troncoso*	1396
Autofundoscopy	*L. C. Drews*	1403
Orthoptic training and surgery for strabismus	*C. Berens, A. J. Elliot, and L. Sobacke*	1418
Exophthalmos	*E. E. Covitz*	1423
Retinochoroiditis of the Jensen type with glaucoma	*T. H. Cowan*	1429
A simplified entoptic pupillometer	*D. G. Cogan*	1431
Apparatus facilitating study by biomicroscopy	*P. Heath*	1433

DEPARTMENTS

Society Proceedings	1434
Editorials	1442
Book Notice	1446
Abstracts	1447
News Items	1473
Index to Volume 24	i

For complete table of contents see advertising page V

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'TWAS THE DAY AFTER CHRISTMAS

'Twas the day after Christmas, and up at the Pole
St. Nick had returned from his gift giving role.
With spirits so merry and heart that was light
He thought of the presents he'd spread through the night.

"These doctors are clever," he chuckled with pride
As he counted the offices he'd been inside.
Why, during the wee hours he'd had to troop
To many a suite with a lens set or loupe.

At professional offices he'd left behind
Refractors, projectors, and things of that kind
As gifts the refractionists knew they would need
In helping their practices thrive and succeed.

"The man who invests in his practice is wise,"
He said with a gleam in his knowing old eyes,
"I've been around and I surely can tell
That he's twice as wise who buys B & L."

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AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 24 DECEMBER, 1941 NUMBER 11

EXPERIMENTAL STUDIES ON VITREOUS DETACHMENT*

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Interest in the problem of vitreous detachment has increased in recent years for two reasons: first, because of the revival of Leber-Nordensen's vitreous theory of retinal detachment in the doctrine of Gonin and in the experimental work of Lindner on the dynamics of detached vitreous; and second, because of the frequency of its occurrence with retinal detachment, with myopia, and with senile changes.

In view of these facts a study of the pathogenesis of vitreous detachment is of practical importance. Explanations and discussions of this question have had a purely hypothetical basis since there has been no experimental, clinical, or anatomic proof of any theory excluding shrinkage of the vitreous due to an organized exudate or loss of vitreous due to a perforating injury.

It is generally assumed that a primary shrinkage or a primary loss of volume of the vitreous body is responsible for the development of a vitreous detachment. This assumption was used as a working hypothesis for the first part of the studies reported here. The experiments represent an attempt to investigate some factors which reduce the volume of the vitreous body *in vitro*.

The technique of the volume determinations of pigs' vitreous was the same as that described in a previous paper[1] and consisted principally in the prevention of an uncontrolled loss of capillary-bound vitreous fluid by leaving the ciliary body and nerve head with surrounding retina in place.

The experiments fall into the following groups:

I. Experiments with protected vitreous preparations
   - A. Effect of H-ion concentration
   - B. Effect of hypertonicity
   - C. Effect of dehydrating neutral salts in high concentrations (extended to experiments *in vivo*)
   - D. Effect of changes in the electric charge
   - E. Effect of pressure
   - F. Effect of enzymes (extended to experiments *in vivo*)

II. Experiments with vitreous preparations without nerve head

Some topics will be treated briefly since they represent a confirmation of known facts.

A. The effect of H-ion concentration

Relative to the effect of H-ion concentration, no loss in volume was found in the range of pH 4 to 11. The irreversible shrinkage beyond this range is without applicability to the living eye.

B. The effect of hypertonicity

As might be expected, hypertonic sur-

* From the Department of Ophthalmology, College of Physicians and Surgeons, Columbia University, and the Institute of Ophthalmology, Presbyterian Hospital. Supported by the Knapp Memorial Foundation. Presented at the twelfth annual meeting of the Association for Research in Ophthalmology, at Cleveland, June 3, 1941.

rounding fluid generally decreased the volume of the vitreous preparations. Such volume changes were reversible. The effect of a slight increase of the osmotic concentration of the outside fluid was, however, overcompensated by a swelling tendency observed in such preparations. Pigs' vitreous began to lose in volume when the concentration of the solution of sodium chloride exceeded 1.2 percent.

It is known that the osmotic concentration of body fluids, if artificially increased, is quickly restored. The results of hypertonicity could be therefore only temporary.

C. The effect of dehydrating neutral salts in high concentrations

The experiments of the next two groups have as their theoretical basis the response of real hydrogels to the action of neutral salts in high and low concentrations. Hofmeister[2] studied the effect of neutral salts on gels and arranged them in a series with regard to their swelling and shrinkage effect. Later the applicability of the Hofmeister series was studied in all fields of biology. The results of experiments on vitreous preparations will be compared with the work of Bungenberg de Jong and his co-workers[3] on gelatin and agar. The explanations of the action of the so-called lyotrope series of anions must be omitted here, but it can be stated briefly that hydration and electric charge of the micelles are supposed to be the factors on which volume changes depend.

The studies reported here are concerned with the dehydrating action of neutral salts in high concentrations. The shrinkage obtained in 0.25, 0.5, and 1 normal solutions of these salts reached 25 percent of the original volume (in 1 N K_2SO_4) and was completely reversible.

The investigations were extended to experiments on rabbits, and vitreous detachment was produced by the intravitreal injection of dehydrating salts with the following technique: An intravitreal injection of 0.2 c.c. of a normal solution of potassium sulphate and other salts of the Hofmeister series was made with a fine needle (27 gauge). To prevent the escape of the injected fluid or of vitreous, the anterior chamber was emptied previously by paracentesis.

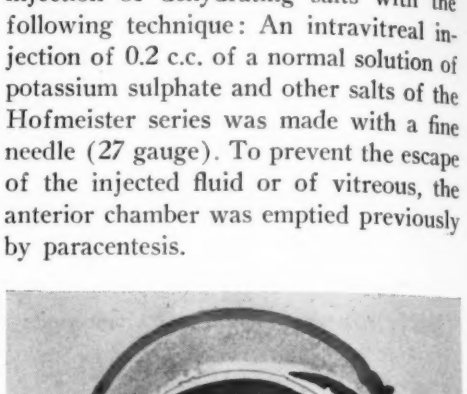

Fig. 1 (Sallmann). Detachment of vitreous after intravitreal injection of 0.2 c.c. of a normal solution of potassium sulphate (Szent-Györgyi fixation).

Vitreous detachment was found 24 hours after the injection of potassium sulphate but was absent in eyes removed after several days and also in eyes injected with equimolar solutions of hydrating salts. It appears therefore that this type of experimental vitreous detachment is reversible—like the shrinkage of the vitreous observed *in vitro*.

To avoid injury by injection, the anion was introduced into the vitreous by iontophoresis in another series of experiments: Under pernoston anesthesia the sclera was exposed by a conjunctival incision at the upper circumference. The superior rectus and superior oblique were displaced by sutures, and a Birkhäuser electrode, 9 mm. in diameter, containing a normal solution of the salt, was placed on the

sclera for 5 minutes at 5 milliamperes. The penetration of the salts into the vitreous was tested qualitatively.

No vitreous detachment was observed by the introduction of the sulphate with this method, but an average 3- to 5-mm. lowering of the intraocular pressure, below the figures obtained with sodium chloride or hydrating salts, was evident for several days. It does not seem improbable that the decrease in intraocular tension was caused by dehydration.

D. The effect of changes in the electric charge by neutral salts in low concentrations

There is a remarkable resemblance between the outcome of experiments on vitreous preparations and experiments reported on gelatin and agar in solutions of neutral salts in low concentrations. The resulting shrinkage is supposed to be a discharge phenomenon depending in its extent on the valence of the ion whose charge is opposite that of the gel. In the negatively charged colloid system of the vitreous at a physiologic pH, the valence of the cation is effective. Desalinated vitreous preparations in equimolar solutions (5 and 10 milliequivalents) of the monovalent potassium chloride showed less shrinkage than in bivalent salts ($BaCl_2$ and $CaCl_2$). The shrinkage in solutions of the trivalent hexamine cobalt chloride surpassed that in solutions of the mono- and bivalent salts. In the experiments with neutral salts, therefore, the vitreous preparations acted like real hydrogels.

E. The effect of pressure

Pressure from the outside is another factor that may reduce the volume of the vitreous. The activity of the external ocular muscles, the intensive contraction of the orbicularis, contusion, and—among the therapeutics—massage, can be regarded as pressure factors. Massage performed on an aphakic eye, the lens having been removed intracapsularly, demonstrates such volume changes clearly. As experiments *in vitro* show, volume losses due to pressure are reversible, if the loss does not exceed 10 percent of the volume.

F. The effect of enzymes

In the last group of experiments with intact vitreous preparations, the influence of enzymatic factors on volume changes was studied. The frequent occurrence of vitreous detachment in early stages of endophthalmitis septica and of thrombosis of the central retinal vein suggested an investigation into the effect of proteases. Numerous experiments were performed with trypsin: it was injected into the preparations; its activity was varied with heat; it was used as the outside fluid in varying dilutions, at different H-ion concentrations, and at different temperatures. These experiments were followed by others on rabbits, and consisted of the intravitreal injection of trypsin solutions. In no way were the results consistent.

The effect of leucoproteases was examined *in vivo* by the intravitreal injection of washed leucocytes produced in the pleural cavity of other rabbits by the aleuronate method. In two instances a beginning vitreous detachment was observed; because of the injury by injection, the results cannot be attributed with certainty to an enzymatic action.

Meyer and his co-workers[4] consider a mucopolysaccharide, hyaluronic acid to be the gel-forming factor of the vitreous. Hyaluronidase hydrolizes this compound.[5] Preparations of pneumococcic and testicular extract containing the enzyme were used to study its effect on vitreous preparations. The volume changes following the injection of 0.2 c.c. of the enzyme into the preparations were compared with those after the injection of the same amount of heat-inactivated enzyme and of a 0.9-percent solution of

sodium chloride. No volume changes occurred that could be ascribed to the activity of the hyaluronidase.

The experiments with enzymes did not, therefore, reveal any convincing relation to shrinkage of the vitreous. But that such a relation does exist cannot be denied in the case of negative results, with the technique used.

Discussion

The experiments on protected vitreous preparations demonstrated that volume losses produced by osmotic and dehydrating processes, by shifts in the electric charge, and by pressure are generally reversible.

These results cannot be applied to the living eye without reservation. Many factors—such as regeneration and exchange of vitreous fluid and other basic biological processes, permeability of the condensation membrane under normal and pathologic conditions, and elasticity of the sclera—cannot be evaluated in their relation to the volume changes of the vitreous. On the other hand, there is little doubt that the factors examined *in vitro* influence the volume changes of the vitreous in life. It is probable that the volume losses produced by these factors *in vivo* are also reversible and transitory, especially if the condensation membrane of the vitreous is not damaged.

II. Experiments with vitreous preparations without nerve head

This leads to experiments in the second part which were performed on vitreous preparations without nerve head, accepting as a working hypothesis the possibility of a vitreous detachment without a primary shrinkage.

A disc-shaped defect of the condensation membrane at the posterior pole is a common finding in cases of myopic and senile vitreous detachment. The defects are thought to be caused by the separation of the vitreous from its insertion at the disc border in the area martegiani.

A comparable result was achieved *in vitro* by cutting off the nerve head and the adjacent retina. The damaged preparations were then suspended in 0.9 percent sodium-chloride solution and in freshly prepared vitreous fluid. Instead of the increase in volume generally found in intact vitreous, a decrease was noted in most of the modified experiments. The escape of the free, capillary-bound fluid at the pos-

TABLE 1

Volume changes of preparations of pigs' vitreous with and without nerve head

With nerve head in vitreous fluid			*Without* nerve head in vitreous fluid		
Original Volume c.c.	Volume after 20 hours c.c.	Percent Change	Original Volume c.c.	Volume after 20 hours c.c.	Percent Change
4.15	4.65	+12.0	5.25	5.05	−3.8
5.02	5.35	+6.55	5.5	4.8	−12.7
5.7	6.05	+6.13	5.0	5.0	0
6.07	5.93	−2.3	6.38	6.25	−2.04
4.8	5.15	+7.27	5.7	5.7	0
With nerve head in 0.9% NaCl			*Without* nerve head in 0.9% NaCl		
4.8	5.25	+9.35	4.95	4.75	−4.3
4.2	4.5	+7.14	4.25	3.95	−7.05
4.7	5.2	+10.6	5.55	5.25	−5.4
4.87	5.15	+5.73	5.58	5.5	−1.43
5.8	6.2	+6.89	4.75	4.74	−2.1

terior pole was the probable cause of the loss in volume.

When the preparations without nerve head were suspended in solutions of strongly hydrating salts, as 0.5 normal potassium thiocyanate, they showed a loss of 7 percent instead of the average swelling of 25 percent.

Returning to clinical cases of vitreous detachment with disc- or ring-shaped defects of the condensation membrane at the posterior pole, it will be remembered that degenerative processes at the nerve entrance in the form of choroidal atrophy are present in many instances. It can be assumed in such cases that degenerative changes around the nerve head involve the structures of the area martegiani and lead to a seepage of free fluid into the subvitreal space, thereby increasing the separation of the involved parts. One or more of the factors mentioned in the first part, generally producing a transitory decrease in volume, may influence the further development of the vitreous detachment. Clinical experience and examination *in vitro* agree with the acceptance of this form of a vitreous detachment as a persistent one; that is, not reversible.

Discussion

In the experiments of other investigators on naked vitreous or parts of vitreous a slow decrease in volume was found in solutions of physiologic salt concentration and in hypotonic solutions. This loss can be attributed to the escape of capillary-bound fluid. The experiments reported here showed that a similar loss of fluid and decrease in volume followed an injury of the condensation membrane at the nerve entrance.

Whatever the physiologic importance of the area martegiani in the process of water transport and exchange may be, the course of a vitreous detachment, its clinical symptoms and signs, favor the concept that a defect of the condensation membrane at the nerve entrance causes the detachment.

Experiments *in vitro* support this theory.

Summary

1. The effects of osmosis, dehydration, discharge, and pressure on volume decrease and the basic reversibility of the loss were demonstrated in experiments on the vitreous *in vitro* and in some experiments *in vivo*.

2. No conclusive results of the effect of enzymes (proteases, hyaluronidase) were observed *in vitro* or *in vivo*.

3. In contrast to the volume increase of protected-vitreous preparations in 0.9-percent sodium chloride, a loss in volume was obtained by damaging the posterior pole of the preparation.

4. The results with damaged-vitreous preparations support the theory that a vitreous detachment may be caused by degenerative changes in the area martegiani.

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GLIOMAS OF THE RETINA*

A HISTOPATHOLOGIC STUDY

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Before their origin was recognized, tumors of the retina were long known as "fungus medullaris" or "fungus hematodes."[37] Wardrop[51] in 1809 gave the first comprehensive clinical description and called attention to the fact that there were "cancers" of the eye which arose solely from the retina. Virchow[50] in 1864 pointed out the presence in the retina of supporting tissues analogous to the neuroglia of the brain, and was the first to attribute to these neuroglial cells the origin of the retinal tumors, for which he therefore proposed the name "glioma of the retina." Following Virchow, numerous workers, including Hirschberg,[25] Knapp,[27] Greeff,[20] and others, reported studies confirming the gliomatous nature of these tumors, and although various other classifications were offered, such as "glia angiosarcomata" (da Gama Pinto,[14] 1886) and small round-cell sarcoma (Delafield,[15] 1871), Virchow's conception of the nervous origin of these tumors eventually gained widespread acceptance, and the term "glioma retinae" came into general use.[37]

In 1891 Flexner[17] first described in a retinal tumor the formation known as a "rosette"; that is, a ring of columnar cells arranged about a central circular lumen, having a fine "limiting membrane" just within the inner border of the cells and sometimes delicate protoplasmic cell processes projecting into the lumen, a picture which suggested to him the form of the rod-and-cone and external-nuclear layers of the retina; that is, the layers formed by the rod-and-cone cells. Tracing the origin of his tumor to a small point in the external-nuclear layer, Flexner concluded that the tumor arose from the rod-and-cone cell layer, the layer designated by Schwalbe as the sensory epithelium or "neuro-epithelium" of the retina. Flexner therefore proposed for this type of tumor the term "neuro-epithelioma."

Grinker[21] (1931) offered a classification of the retinal tumors that divided them into three groups: (1) medulloepitheliomas, consisting of neuro-epithelium, retinoblasts, and primitive retinal epithelium, and arising from persistent primitive retinal epithelium in the pars ciliaris retinae, or ciliary epithelium; (2) neuro-epitheliomas, consisting of spongioblasts in the form of rosettes, as well as primitive spongioblasts, astroblasts, astrocytes, and probably oligodendroglia, and arising from primitive spongioblasts or neuro-epithelium; and (3) retinoblastomas, the most common type, arising from and composed of small undifferentiated cells of the type called "retinoblast," a hypothetical bipotential embryonic cell assumed to be derived from the germinal cells of the primitive retinal epithelium.

In 1937 McLean[31] reported a tumor arising in the inner layers of the retina which he classified as an astrocytoma. No such tumor was found in our material, but we had the privilege of examining a section of McLean's specimen (fig. 1). The tumor was composed chiefly of spindle-shaped cells with long fibrillary processes, which McLean showed by differential stains to be neuroglial tissue. This appears

* From the Section on Surgical Pathology and the Section on Ophthalmology of the Mayo Clinic. Read at the twelfth annual meeting of the Association for Research in Ophthalmology, Cleveland, Ohio, June 3, 1941.

to be a rare if not unique case of retinal tumor composed of well-differentiated glial cells, although Verhoeff[46] and others had predicted the possibility of such an occurrence.

In an effort to determine the nature of the retinal tumors that we have seen and to sort them into a useful classification, we have made a histopathologic study of 35 eyes surgically removed because of retinal

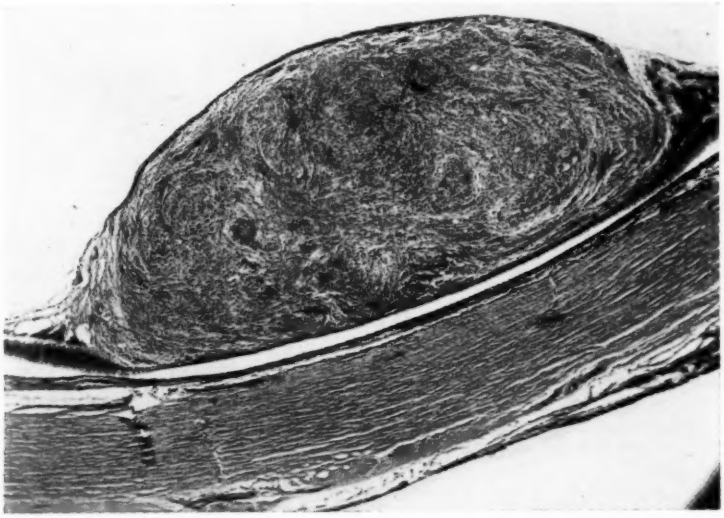

Fig. 1 (Parkhill and Benedict). Astrocytoma, case reported by McLean. The tumor arises in the inner layers of the retina (×18).

tumor. Sections stained with hematoxylin and eosin were examined in every case, and where material was available for special stains, additional techniques were employed. These included Mallory's phosphotungstic-acid hematoxylin for glia fibrils, Weigert's elastic-tissue stain, van Gieson stain for connective tissue, and cresyl violet for ganglion cells. Heavy-metal-impregnation methods included the Bielschowsky procedure for nerve cells and processes and the modification suggested by Orlandi; the Bodian technique for nerve cells and processes; Cajal's gold-chloride and sublimate technique for astrocytes and processes, and Hortega's silver-carbonate method for microglia and oligodendroglia.

Of the 35 patients from whom eyes were removed, all but one had been examined at the Mayo Clinic; in three cases, the eye was removed elsewhere and tissue or sections were sent to us for our examination. Clinical data on the 35 cases are summarized in table 1. In 15 cases the left eye was involved and in 15 the right; in four of the 35 cases (11.4 percent) both eyes were involved. In one case the situation was not recorded. In a series of 405 collected cases quoted by Parsons, the right eye was involved in 143 cases and the left in 165; both right and left eyes were involved in 97, or almost 24 percent, a figure which represents the incidence of bilateral involvement generally quoted. Eighteen of our cases were in males, 16 in females; in one, the sex was not recorded. In a series of 704 cases collected by Owen, 346 were in males, 327 in females, and 31 not recorded.[36]

Grossly the retinal tumors were grayish or whitish, nodular, and fairly well cir-

TABLE 1

CLINICAL DATA ON CASES OF RETINAL GLIOMA

Case	Sex	Eye	Location Primary Tumor	Extraocular Extension	Age at Operation, years	Duration Symptoms before Operation, years	Results, Living or Dead	Duration of Life after Operation	Recurrence		Type of Tumor	Remarks
									Interval after Operation	Site		
1	M	Rt.	Post.?	Optic nerve	4	2	Died	Not known	8 mo.	Orbit?	Ret.*	
2	M	Rt.	Diffuse	Optic nerve; orbit	3	9/12	Died	19 da.	?	?	Ret.	Glioma lt. eye
3	F	Rt.	Post.?	Optic nerve; episcleral	4	1 1/12	Recurrence	?	5 mo.	Orbit; meninges?	Ret.	Final outcome not reported
4	F	Rt.	Diffuse	Optic nerve; episcleral	4	1 2/12	Died	21 da.	?	?	Ret.	Convulsions 2 days postop.
5	F	Lt.	Post.	Optic nerve	4	10/12	Died	5 mo.	5 mo.	Orbit; meninges	Ret.	Twin sister (case 32) had bilat. gliomas
6	F	Rt.	Diffuse	Optic nerve	2 1/12	7/12	Died	9 mo.	Prob. cranial sion at mission	intra- exten- 1st ad-	Ret.	Comatose on admission; left eye removed 4 mo. after rt.
7	M	Rt.	Diffuse	Optic nerve; episcleral	1 4/12	8/12	Died	5–6 mo.	?	C.N.S. ?	Ret.	Glioma lt. eye; radium treatment
8	M	Lt.	Post.?	Optic nerve	2 6/12	7/12	Died	10½ mo.	7 mo.	Orbit; C.N.S.?	Ret.	Convulsions 9 mo. postop.
9	F	Lt.	Post.?	Optic nerve; episcleral?	2 4/12	1 2/12	Died	less than 9 mo.	?	?	Ret.	
10	M	Lt.	Diffuse	Optic nerve; episcleral	6 4/12	1	L & W†	14 yr.	3½ mo.	Orbit	Ret.	Recurrence; radium treatment
11	M	Lt.	Diffuse	Orbit; optic nerve	4	1 5/12	Died	5 mo.	?	?	Ret.	Elevated retina. Rt. eye seen 3 mo. postop.; shrunken globe
12	M	Lt.	Diffuse	Optic nerve; episcleral?	2 6/12	1	Died	Probably about 4 mo.	3 mo.	Intracranial	Ret.	
13	F	Lt.	Diffuse	Optic nerve; orbit	2 6/12	9/12	Died	7 mo.	3 mo.	Orbit; preaur.	Ret.	
14	?	?	Post.	Optic nerve	3	?	?	?			Ret.	Patient not seen at clinic; slide sent in
15	M	Rt.		Episcleral?	4	2 da.	Died	1 yr., 3 mo.	4 mo.	Orbit; lid	Ret.	3 rec., treat. with radium and excision
16	F	Lt.	Post.?	—	1	16 da.	L & W	13 yrs.	—	—	Ret.	
17	F	Rt.	Diffuse	—	1 9/12	2/12	Prob. L & W 6 yrs.				Ret.; phth. bulbi	Strabismus at birth. Letter 6 yrs. after oper. from mother; no mention of recurrence
18	M	Lt.	Diffuse	—	1 6/12	1 3/12	Died	11 mo.	8–9 mo.	Head? arm?	Ret.; phth. bulbi	"Sarcoma—arm; great bumps in head." No recurrence in socket
19	M	Rt.	Anterior	—	3	3/12	L & W	22 yrs.	—	—	Neuroepith.	
20	M	Rt.	Post. & temp.	Optic nerve	2 6/12	1½ mo.	Died	5 wk.	4 wk.	Orbit	Neuroepith.	
21	M	Rt.	Post. & temp.	—	2 6/12	2 wk.	L & W	At least 2½ yrs. Not traced since	—	—	Neuroepith.	

TABLE 1—*Continued*

Case	Sex	Eye	Location Primary Tumor	Extraocular Extension	Age at Operation, years	Duration Symptoms before Operation, years	Results, Living or Dead	Duration of Life after Operation	Recurrence		Type of Tumor	Remarks
									Interval after Operation	Site		
22	F	Lt.	Post. & diffuse	—	3+	2½ mo.	L & W	8 yrs.?	—	—	Neuro-epith.	Letter from mother 8 yrs. post-op.; no mention of recurrence
23	M	Rt.	Post.?	—	2 2/12	1 8/12	Died	1 yr.	10 mo.	Humerus; head	Neuro-epith.	X-ray & radium 5–6 mo. before enucleation. Tumor lt. eye treated with radium
24	F	Rt.	Ant.?	—	3	6 mo.	Died	10 yrs.	—	—	Neuro-epith.	No recurrence nor metastasis; died "heart trouble"
25	F	Rt.	Ant.	—	1 2/12	5 da.	L & W	5 yrs.	—	—	Neuro-epith.	
26	M	Rt.	Post.	—	2 6/12	1 mo.	L & W	1 1/12 yrs.	—	—	Neuro-epith.	
27	M	Rt.	Post. & temp.?	—	6/12	2 mo.	L & W	5 yrs.	—	—	Neuro-epith.	
28	F	Lt.	Post. & temp.	—	7/12	2 mo.	?	?	—	—	Neuro-epith.	Not traced
29	M	Lt.	Post.	Optic nerve	3 6/12	4 mo.	Died	6 mo.	3½ mo.	Orbit	Neuro-epith.	Recurrence treated with radium
30	M	Rt.	Diffuse	Optic nerve; episcleral	5	9 mo.	Died	"few weeks"	?	?	Neuro-epith.	Date and site recurrence not given
31	F	Lt.	Post.	—	3 6/12	6? mo.	Died	About 14 mo.	11 mo.	Head	Neuro-epith.	
32	F	Lt.	Ant.	—	4	4 mo.	L & W	18½ yrs.	—	—	Neuro-epith.	Glioma right eye treated with radium. Twin (case 5) died of glioma
33	F	Rt.	Post.	—	2	6 mo.	L & W	9½ yrs.	—	—	Neuro-epith.	
34	M	Lt.	Diffuse	Episcleral	3	9 mo.	L & W	5 yrs.	?	?	Neuro-epith.	Symptoms of tumor returning 2 yrs. postop. X-ray treatment. Enucleation elsewhere
35	F	Lt.	Diffuse	Optic nerve; episcleral	4	2 yrs.	Died	4½ mo.	2½ mo.	Occip.; neck; axilla	Neuro-epith.	

* Ret.—Retinoblastoma type.
† L & W—Living and well.

cumscribed in earlier stages; diffusely filling the vitreous chamber or practically the entire globe in advanced stages. The tumors were soft and friable, at times almost fluid. On cut section small red points representing blood vessels and sometimes small hemorrhages were seen. There were yellowish regions of degeneration, and usually small gritty chalky-white particles of calcium.

The tumors were prone to extend into the optic nerve; within the globe, extension to the choroid was most common and in many cases extensive. The iris, ciliary body, sclera, cornea, and other ocular structures also were involved at times.

Microscopically the characteristic structure was the often-described perivascular arrangement of bands or cuffs of closely packed small cells around tumor blood

vessels; these, in turn, surrounded by a mantle of degenerating and necrotic cells. To account for this characteristic architecture, the explanation has been offered that the rapidly growing tumor cells soon outdistance their blood supply and become degenerated, leaving only a band of living tumor cells adjacent to blood vessels.[37] This, however, is not the whole story, for the same formation occurs in small and probably early tumors; in fact, Parsons said that the perivascular arrangement is invariably present in the early stages. Our study confirms this finding, for such a structure has been found in all the cases in this series, whether the tumors were large or small, of long or short duration. This form was, however, much more extensive in certain instances than in others; in the primary tumors, or tumors still within the vitreous chamber, it was generally the predominating arrangement, whereas in choroidal and extraocular extensions there was usually much less degeneration and, when present, it was distributed more irregularly. It has been suggested that the pressure of the rapid growth of the tumor within the unyielding walls of the globe may be the factor responsible for the frequent degeneration of cells distant from the blood vessels, yet choroidal extensions still confined within the sclera and presumably subject to the same tensions usually show relatively little degeneration of this type. It would appear that the complete explanation remains yet to be discovered. In smaller regions of the primary tumors and in the larger part of intraocular and orbital extensions, the predominating type of growth was usually in relatively solid masses or strands and cords of cells, with relatively small and irregular regions of degeneration.

Various stages of degeneration were seen in the primary tumor, from cells in which the nuclei took the basic stain a little less intensely than normal to those in which the nuclei stained only with acid stains or not at all. In some cells the nuclear chromatin was massed in heavy clumps; in others the nuclei were fragmented and simulated polymorphonuclear leukocytes. Within the islands of degenerated cells, characteristic regions of calcification often were found (in all but five of our tumors) and in some cases there was also a delicate line of calcification in the walls of tumor blood vessels.

The cellular structure most commonly found was that of masses of small cells with scanty, poorly staining, almost invisible cytoplasm, and relatively large nuclei with abundant chromatin staining deeply with hematoxylin. Occasionally a fine tail-like process was seen extending away from one pole of the cell. The cells were for the most part fairly uniform in size, and under the low power of the microscope the nuclei appeared nearly round or spherical, so that the appearance of lymphosarcoma was suggested to some extent. However, under higher power the majority of the nuclei were seen to have a slightly angular or polygonal appearance, or even a form often described as "carrot" or "wedge" shaped.

The cells appeared to be extremely undifferentiated or anaplastic. With special stains for glial and nerve fibrils, no cell processes nor fibrils were demonstrated in these cells, which appeared to be too "primitive" or anaplastic to have developed such evidences of specialized functional differentiation. Such cells are likened by Bailey and Cushing[4] and others to the "retinoblast," a hypothetical highly undifferentiated cell of the developing embryonic retina, comparable to the hypothetical primitive "medulloblast" of the

central nervous system, and like it having the potentiality of differentiating along either neuroblastic or spongioblastic lines. A tumor composed chiefly of such undifferentiated cells is called by Bailey and Cushing and by Grinker[22] a "retinoblastoma." These tumors closely resemble histologically the very cellular, highly malignant tumors of the central nervous system known as "medulloblastomas" and the similar tumors of the sympathetic nervous system called by Wright[53] "neuroblastomas," and they are considered generally to be similar in nature.[4]

All our tumors were found to contain highly undifferentiated cells of the type which Grinker likens to the "retinoblast." Eighteen of our tumors were composed almost entirely of such cells and were of the type called by Grinker[22] and by Bailey and Cushing[4] "retinoblastomas" (fig. 2).

Twelve tumors, while possessing cells of this type, showed in addition a varying amount of rosette formation, and were of the type called by Flexner, Grinker,[21] Bailey and Cushing,[4] and others, neuroepitheliomas (fig. 3). The rosettes were composed of a ring of cuboidal or cylindrical cells arranged about a central lumen with delicate limiting membrane, most of the cytoplasm lying between the polygonal or wedge-shaped, deeply staining nuclei and the inner border of the cells. The rosettes had no external nor basement membrane; long processes tapering away from the base of the cells occasionally were seen. Bailey and Cushing and others have called these cells primitive spongioblasts.

In our cases, rosettes were present in

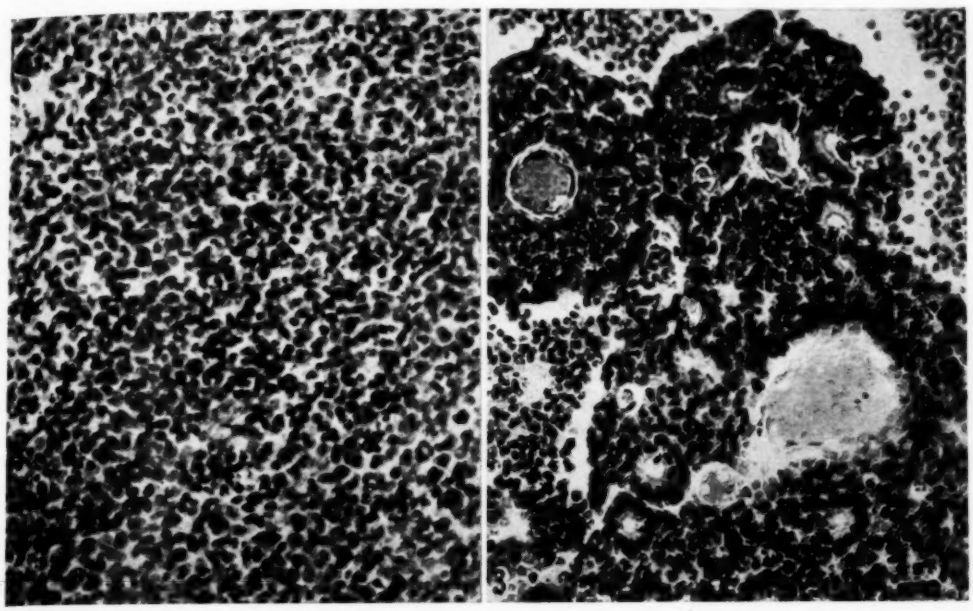

Fig. 2 (Parkhill and Benedict). Case 10. Retinoblastoma type of tumor. Small, extremely undifferentiated cells, with scanty cytoplasm and hyperchromatic nuclei (×210).

Fig. 3 (Parkhill and Benedict). Case 29. Neuro-epithelioma type of glioma. Many well-formed rosettes as well as pseudorosettes are seen. Mitotic figures are numerous (×200).

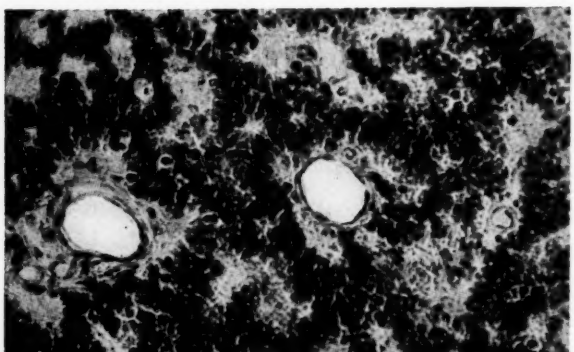

Fig. 4 (Parkhill and Benedict). Case 24. Neuroepithelioma type of glioma. Numerous pseudorosettes. The clear spaces filled with fibrillary material are unusually large (×200).

very small numbers in some of the tumors; in others they were more numerous, and in three they were the predominating structure. A tumor in which rosette formation is seen throughout is rare, though Grinker[22] said a "pure" form may occur arising from the pars ciliaris retinae. Even where few rosettes were present, however, there was usually evident in the remainder of the tumor a marked tendency of the cells to be grouped or "clumped" in short rows or small segments of circles about clear spaces filled with delicate fibrillary material. Sometimes the cells surrounding the clear region were arranged in nearly circular formation, composing so-called pseudorosettes (fig. 4). While these formations usually appeared in abundance in tumors displaying true rosettes, they also were numerous in certain cases (five in our series) in which rosettes were not discovered. The pseudorosette formation suggested strongly an incomplete or partially successful step toward rosette formation; indeed in some of the pseudorosettes regions could be seen in which adjoining cells had developed a "limiting membrane" which, however, did not extend in a complete circle or enclose a lumen. These tumors resembled so closely in structure, origin, and development the tumors with true rosettes that they were considered to be of nearly if not quite the same degree of differentiation and to fall into the same group.

Ocasionally seen were regions in which elongated cells with narrow angular nuclei were arranged in a ring surrounding a blood vessel, the cells packed closely side by side with long axes parallel to each other and perpendicular to the vessel wall (fig. 5). Usually there was little cytoplasm between the nucleus and the inner border of the cell; occasionally an indistinct process was seen extending toward the wall of the blood vessel. When such a vessel was sectioned at right angles,

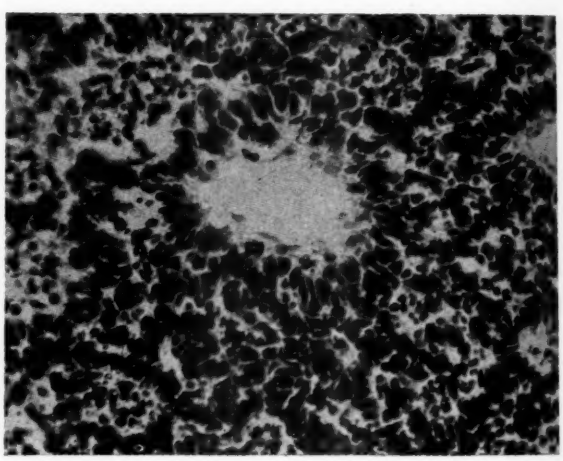

Fig. 5 (Parkhill and Benedict). Case 35. Rings of tumor cells surrounding blood vessels; also bands of tumor cells surrounding small areas of degeneration; sometimes called pseudorosettes (×275).

a picture imitating a rosette was seen. Sometimes, in fact, such structures have been described as true rosettes. Such an arrangement is sometimes present in medulloblastomas of the central nervous system. It was seen in five of our tumors, in all of which either the rosette or pseudorosette structure predominated. The cells had somewhat the appearance of epithelial cells and, we believe, were also evidences of slight differentiation in the tumor. The alignment of tumor cells in a ring about a blood vessel often is referred to as a pseudorosette. Another arrangement frequently described as a pseudorosette is that resulting from the degeneration of a small group of tumor cells leaving a clear space, generally containing some cellular débris, surrounded by a ring of living cells. However, these forms are not considered here to be pseudorosettes; this term is reserved for the previously described structural development of irregular circles or segments of circles of cells surrounding a clear region containing fibrillary material.

A variation of the rosette structure rarely seen was in the form of narrow rows of elongated angular cells, sometimes with a distinct limiting membrane bordering a narrow canal. The cells were aligned with their long axes parallel to each other and perpendicular to the limiting membrane surrounding the canal (fig. 6). This arrangement resembles that seen in the rare medullo-epitheliomas of the central nervous system. It appeared in none of our tumors as the predominating cellular structure, and in small regions in only two. We had no tumors in our group of the type classified by Grinker[22] as medullo-epithelioma. In his cases, collected from the literature (Badal and Lagrange,[2] Coats,[10] Collins,[11] Cucchia,[13] Emanuel,[16] Knapp,[27] Kuthe and Ginsberg,[29] Leber,[30] Meller,[33] Redslob,[41] Schlipp,[44] Velhagen,[45] and Verhoeff[47]), the tumors arose in almost every instance from the epithelium of the ciliary body and were differentiated to form glandular canals and in some cases even primitive optic vesicles. From the descriptions given by the various authors, one would conclude that the differentiation in many

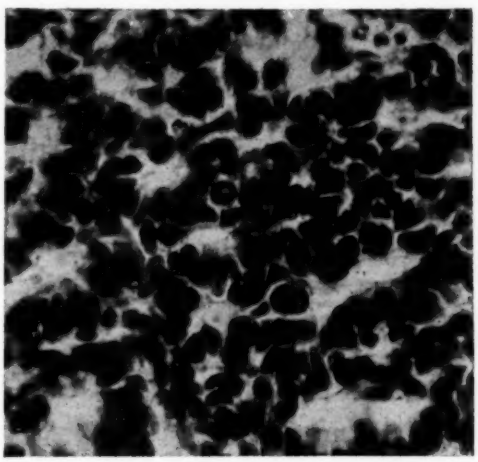

Fig. 6 (Parkhill and Benedict). Case 7. Tumor cells with elongated angular nuclei, surrounding narrow irregular canals. Tripolar mitotic figure slightly to right of center (×360).

of these tumors was relatively advanced, even to the formation of vitreous in Verhoeff's case, and that the tumors were far less malignant than the rare, extremely undifferentiated medullo-epitheliomas of the central nervous system. The fact that the cases reported were usually of long duration (for example, Badal and Lagrange, 8 years; Redslob, 11 years; Emanuel, 5½ years) would tend to bear out the conclusion that these are relatively well-differentiated tumors of comparatively slow growth and low grade of malignancy.

In all our tumors mitotic figures were present and usually abundant. They were rare or absent in degenerating regions but occurred often in large numbers in areas of well-preserved, actively proliferating cells. Mitotic figures were extremely num-

erous in most of the regions of rosette formation (fig. 3) as well as in some of the highly undifferentiated cell masses. In many of the latter regions, however, especially when degeneration was marked, mitotic figures were found less frequently. A few atypical mitotic figures were seen; one showed well-marked tripolar division (fig. 6).

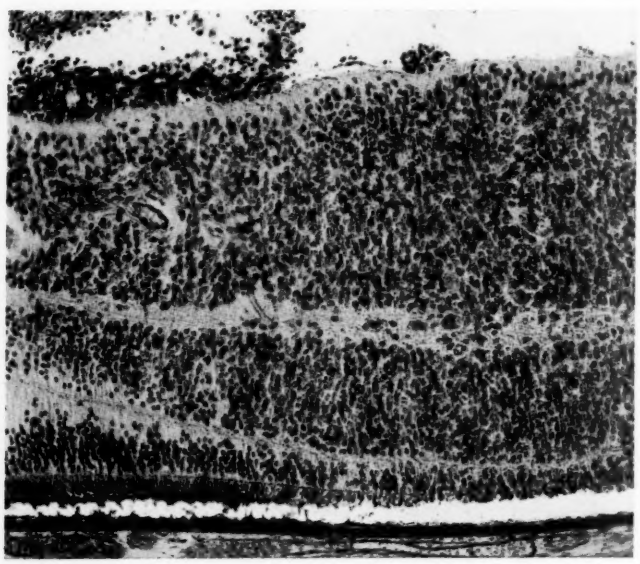

Fig. 7 (Parkhill and Benedict). Case 34. Markedly thickened ganglion-cell and inner-nuclear layers, replaced by tumor cells. The appearance is that of tumor transformation *in situ* in these layers (×115).

In all but two of the 17 cases of tumor with rosette or well-marked pseudorosette formation, regions of uninvolved retinal tissue were still present. These regions varied in extent, and eventually became converted into or invaded by tumor. There were, in many cases, regions of normal retina alternating with regions replaced in whole or in part by tumor cells; the appearance was that of tumor of multicentric origin. In a number of cases tumor cells could be seen apparently arising in one or more of the retinal layers; most often in the inner-nuclear layer; frequently also in the ganglion-cell and nerve-fiber layers; or simultaneously in two or more layers (fig. 7). The picture most often seen was a progressive rapid widening or thickening of the retinal layer and replacement of the normal cells by malignant cells (fig. 8). The appearance was that of a malignant transformation *in situ*. In many other instances the tumor cells could be seen in the nuclear or ganglion-cell and nerve-fiber layers, but with a more irregular arrangement, making it difficult to determine whether the tumor was originating in these layers or invading them from another part.

In the outer-nuclear layer a different and very interesting picture usually appeared. The outer-nuclear layer often remained uninvolved over a considerable region in which the inner layers already were replaced by malignant cells (fig. 9). In many places the outer-nuclear layer was degenerated and thinned, sometimes to a single layer of cell nuclei; it appeared as if compressed by the expanding tumor in the other layers. Occasionally tumor cells were found in the external nuclear layer but they usually gave the appearance of being an invasion or extension from elsewhere. At times the inner half of the layer was occupied by tumor cells, while the outer half, usually quite distinctly demarcated, still retained several rows of normal rod-and-cone nuclei, bounded by the external limiting membrane, and often accompanied by persisting, though more or less degenerate, rods and cones. In only one or two instances was there an appearance definitely suggestive of tumor arising *in situ*

in the outer-nuclear layer (fig. 7), and even in these cases the possibility of invasion from other layers could not be ruled out definitely.

These findings are contrary to the original conclusions reported by Flexner and the statements of various authors, in which the origin of neuro-epitheliomas is attributed to the outer-nuclear layer. Others, including Wintersteiner[52] and Parsons, on the other hand, stated that while the tumors may arise in various retinal layers they arise most often in the inner-nuclear layer. This is in accord with our observations; we found the tumor apparently definitely arising in the inner-nuclear layer in seven cases, in the ganglion-cell and nerve-fiber layers also in three cases; in one case the origin appeared to be chiefly in the ganglion-cell layer (fig. 10). In many other instances tumor cells could be seen in various retinal layers, but with a more irregular arrangement suggesting the probability of invasion from elsewhere rather than origin *in situ*.

In almost all the cases there was some degree of extension of the tumor within the globe. Of the 17 tumors with rosette or pseudorosette formation, 11 had invaded intraocular structures. This invasion was for the most part slight, such as a few clumps of tumor cells lying free in the posterior or anterior chambers, or a small group of cells growing between the retinal pigment epithelium and the lamina vitrea of the choroid. In only four cases of this group was there extensive invasion of the choroid; in three of these there was also invasion

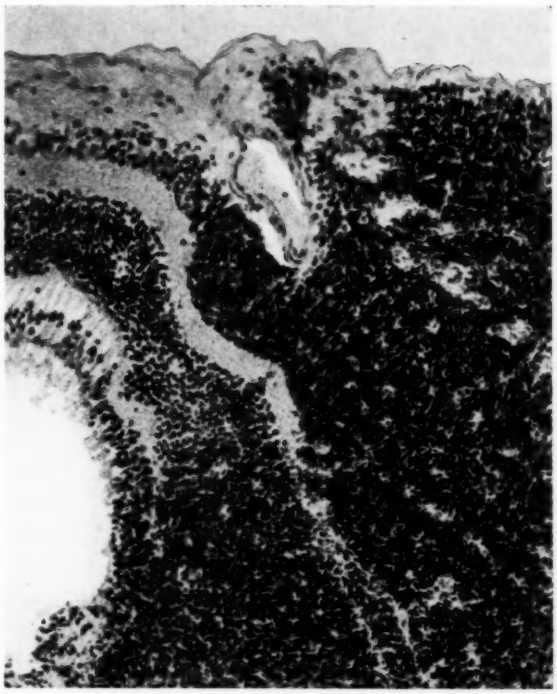

Fig. 8 (Parkhill and Benedict). Case 21. Normal ganglion-cell and inner-nuclear layers (left) becoming markedly thickened by tumor cells (right and below) (×145).

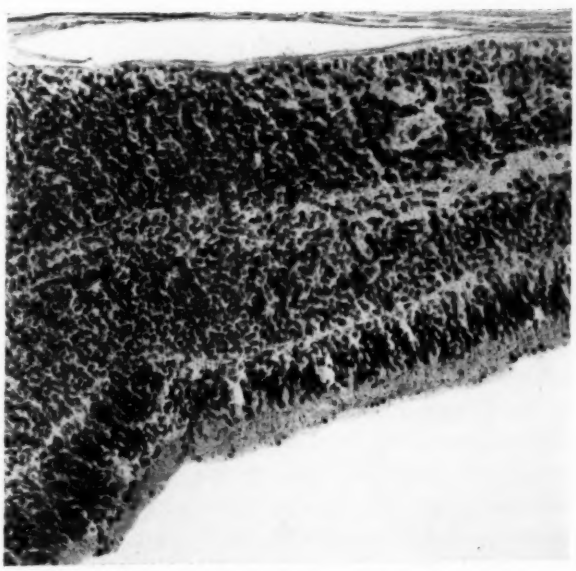

Fig. 9 (Parkhill and Benedict). Case 27. Tumor *in situ* in ganglion-cell and inner-nuclear layers; beginning invasion of outer nuclear layer (lower left) (×160).

of the optic nerve. In only one was there extensive orbital involvement, and in this instance the primary tumor was large and made up of undifferentiated cells; the only region showing rosette formation was in a small extension of the tumor on the anterior surface of the iris.

Six of the patients who had tumors classified as of the neuro-epithelioma type

Fig. 10 (Parkhill and Benedict). Case 28. Glioma of neuro-epithelioma type apparently arising in ganglion-cell layer (above). Below, the normal retina is folded on itself in a hairpin curve (×200).

(cases 20, 23, 29, 30, 31, 35) are known to have died as the result of tumor, at intervals ranging from five weeks to one year after operation. One of these patients had bilateral tumors, the tumor in the second eye being treated by radium only; the tumor in the enucleated eye also had been treated by roentgen and radium therapy at intervals for 20 months before it was removed. In four of these cases there was extension to the optic nerve at the time of enucleation. The duration of symptoms before operation in the fatal cases varied from one-and-a-half months to two years. One other patient in this group died at the age of 13 years, of "heart trouble," 10 years after enucleation of the eye, with no evidence of recurrence, new tumor, or metastasis. In this case the result was considered to have been good so far as the glioma was concerned.

One patient was not traced after operation; the remaining nine patients in this group have been reported living and well, for a duration of from 1 to 18 years after operation; the duration of symptoms before operation in these cases varied from two weeks to nine months.

The patient reported living 18 years after operation (case 32) is interesting in that she survived bilateral retinal tumors. The left eye, containing the larger tumor, was enucleated; the smaller tumor in the right eye was treated with radium, with subsequent ophthalmoscopic evidence of complete regression of the tumor. The right eye was removed elsewhere 11 years later because of inflammation and phthisis bulbi; unfortunately the specimen could not be secured for microscopic study. This case is of further interest as the only one in our series with a history of familial involvement. The patient's twin sister (case 5) died at four years of age as the result of glioma of the left eye. These two cases have been reported previously by one of us (Benedict[5]).*

Of the 18 cases of tumor of retino-

* Since the present study was completed, the daughter of this patient, aged two-and-a-half years, has been brought to the clinic (January, 1941) with gliomas present in both eyes. Details of this case will be reported elsewhere.

blastoma type, only four retained uninvolved retina in the eye (cases 2, 14, 15, 16). Tumor cells were found in the ganglion-cell layer in all four, in the inner-nuclear layer in three, and in the outer-nuclear layer in two cases; but in almost every instance the appearance suggested invasion from elsewhere rather than tumor formation *in situ*.

The optic nerve was invaded by tumor in 14 of the 18 cases in this group. In all there was invasion, usually extensive, of intraocular structures, and in 11 cases extraocular extension was present.

In only 2 (cases 10 and 16) of the 18 cases was the patient known to be living and well, one for 13 years and one for 14 years after operation. In case 16 the eye had been removed 16 days after the tumor was first discovered, and was one of the three in which no extension to optic nerve nor orbit was found. Case 10 was remarkable in that the tumor was highly undifferentiated, with episcleral and optic-nerve involvement at the time of enucleation, and recurrence in the orbit three months after operation, which was treated with radium, resulting in a 14-year cure. This is the only case in our series in which a patient with optic-nerve involvement recovered.

Two of the 18 patients in the retinoblastoma group were not traced; another showed recurrence in the orbit and probable meningeal involvement four-and-a-half months after operation; but the final outcome was not reported. In the remaining 13 cases the outcome is known to have been fatal from 19 days to 11 months after operation.

The only other two specimens in this group without evident optic-nerve or extraocular involvement were from two very interesting cases of glioma in eyes showing atrophy or "phthisis bulbi" (cases 17 and 18). There was no history nor evidence of rupture of the globe in either case; in case 18 there was a history of inflammatory reaction in the eye, followed by shrinking, 14 months previous to enucleation (fig. 11). In both cases, there was seen on microscopic examination a typical shrunken globe, with increase in intraocular fibrous tissue, demonstrated by van Gieson and Mallory's phosphotungstic-

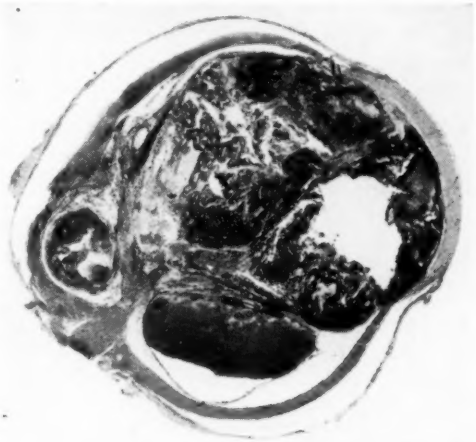

Fig. 11 (Parkhill and Benedict). Case 18. Phthisis bulbi associated with glioma. The globe is shrunken and bands of fibrous tissue extend forward from the optic disc; a hemorrhagic degenerating tumor mass fills most of the remaining vitreous chamber and extends forward to the cornea (×3).

acid hematoxylin stains to be chiefly connective rather than glial tissue. No normal retinal tissue could be found, and the distorted vitreous cavity was filled with extremely degenerated tumor cells; in case 18, invasion of the choroid and of the anterior chamber could be seen. Even though there was marked degeneration of the tumor, a perivascular arrangement of cells was still discernible, with regions of calcification in degenerated cells distant from the blood vessels. The proportion of living tumor cells was not large, but the cells were characteristic in appearance, and showed as many as three to six mitotic figures per high-power field in the best

preserved areas. These two cases are of especial interest in that glioma retinae associated with phthisis bulbi rarely has been reported, and it has even been questioned whether phthisis bulbi ever occurs in a gliomatous eye.[37]

Comment

Since Virchow in 1864 first asserted that retinal tumors arise from glial cells of the retina, there has been much controversy concerning their origin and development. In the earlier years of the discussion the most popular hypothesis was that the tumors arise from misplaced embryonic cell rests, either glial cells or rod-and-cone cells. Even in recent times it has been said that retinal tumors arise from "primitive retinal epithelium."[22]

The supposed resemblance of rosettes to rods and cones has been noted frequently ever since Flexner's original report (1891), and referred to in attempts to explain the origin of retinal tumors. Flexner thought the rosettes were reproducing the rod-and-cone cell layer of the retina, and also traced the origin of his tumor to the outer-nuclear, or rod-and-cone cell, layer.[17] However, his illustration shows not only the outer-nuclear but all the other retinal layers in the involved region replaced by tumor, with no evidence as to which layer or layers were the site of origin.

Wintersteiner in 1894 also observed rosette formations which, he believed, represented rod-and-cone cells, and since no neuroglia had been found in the outer-nuclear, or rod-and-cone cell, layers, he asserted that the tumors could not be of glial origin and that "glioma" was an incorrect term. He suggested that the retinal tumors should be called "neuro-epitheliomas," since he believed that they arose from rod-and-cone cells. To explain the fact that he found tumors arising in various retinal layers, most often in the inner-nuclear layer, he asserted that the tumors arose from embryonic rod-and-cone cells misplaced in other retinal layers.

Pusey[39] in 1902 contended that the tumors were formed, not from the highly differentiated sensory rod-and-cone cells, but from far less highly specialized glia cells; the rosettes, he believed, were formed from neuroglia cells corresponding to the fibers of Müller. Ginsberg concluded that rosettes were not specialized neural epithelium like the rod-and-cone cells, but rather undifferentiated embryonic cells representing the germ of the tumor.[18] Bailey and Cushing pointed out that this did not explain the fact that rosettes are not found in all early tumors, and that they are found sometimes in metastatic growths; they concluded that the cells forming the rosettes are primitive spongioblasts, comparable to the cells of Müller and perhaps at times to embryonic rod-and-cone cells. Munoz Urra[34] in 1923, with tannin silver stains, found various types of cells, many definitely glial cells, and concluded that the tumor cells were glial and not nervous cells. He advanced the novel idea that rosettes are formed by tumor cells arranged between the radiating processes of an astrocyte; the apparent lumen, he claimed, was the body of an astrocyte, not stained by ordinary methods.

Marchesani[32] in 1930, with newer glial stains, found what he called primitive neuro-epithelial cells predominating, some of which differentiated to form rosettes. He expressed the belief that these represent, not the highly specialized rod-and-cone cells, but structures comparable to the primitive neural tube. He also found some "embryonic glial cells" corresponding to primitive spongioblasts, astroblasts, and astrocytes, observing that in many tumors the most differentiated cell was a primitive spongioblast.

In the speculations and investigations regarding the nature of the rosettes and the origin of the tumors, one source of confusion seems to have been the superficial resemblance of the rosettes, with their limiting membrane and projecting cytoplasmic processes, to the rod-and-cone cells, whose outer processes, the rods and cones, project beyond the external limiting membrane of the retina. However, similar rosettes occur in neuro-epitheliomas of the brain,[9] of the spinal cord,[4] and even of peripheral nerves,[38] and rosettes in these tumors are never likened to rod-and-cone cells. It seems more probable that the rosettes represent a partial differentiation of the tumor cells to a form imitating more or less closely the primitive epithelium of the neural tube rather than to the highly specialized rod-and-cone cells.

To explain the origin of retinal tumors, of either the neuro-epithelioma or the retinoblastoma type, it does not seem necessary to assume the presence of embryonic cell rests or of persistent primitive epithelium. It is, in fact, a question whether any truly primitive or embryonic cells persist in fully developed adult tissues or organs. The simple columnar cells of the ciliary retinal epithelium are indeed much less complex in structure and function than are many cells in the visual portion of the retina, but they are, nevertheless, fully differentiated cells, of a relatively simple functional type. To account for the origin as well as the variations in structure and cytologic character of retinal tumors, we may consider the origin of malignant neoplasms in general.

Hansemann has[23, 24] presented a clear conception of the origin of cancers from normal cells, which has been emphasized repeatedly by Broders.[6, 7, 8] By a process of dedifferentiation, or what Hansemann called anaplasia ("backward development"), the previously normal cell changes regressively to produce an anaplastic, undifferentiated cell with decreased or absent specialized functions and, usually, increased reproductive function. Such a change has been produced repeatedly in experimental cancers, where specific carcinogenic hydrocarbons have been applied to normal tissues of experimental animals, followed by the formation of malignant tumors in the treated region, where formerly the cells were normal.[12, 26] As Hansemann[24] and Broders[6] have pointed out, the cancer cell is not an embryonic cell, but an entirely new type, a tumor cell, and may be less differentiated than even the normal embryonic cell. Although tumor cells frequently may be partially differentiated, and often more or less closely reproduce the form of embryonic cells in various stages of differentiation, they do not obey the laws of orderly growth and development toward an always more differentiated type as do embryonic cells, and they may assume many bizarre forms that do not correspond to any normal stage of embryonic development.

It is on the variations in differentiation of tumor cells that Broders bases his grading of cancer and other malignant tumors.[6, 7, 8] The tumors showing the greatest differentiation, or tendency to produce cells like the normal cells from which they spring, are classed as the least malignant, whereas those that show the greatest deviation from the normal differentiated cells are classed as the most malignant. Cancers are graded on a basis of 1 to 4, a grade-1 cancer being one in which differentiation ranges from almost 100 percent to 75 percent; a grade-2 cancer, one in which differentiation ranges from 75 percent to 50 percent; a grade-3 cancer, one in which differentiation ranges from 50 percent to 25 percent; and grade-4 cancer, one in which differentiation ranges from 25 percent to practically none. Conversely, the relative amount of dediffer-

entiation or undifferentiation in the grade-1 cancers is low (ranging from almost none to 25 percent) and increases with each grade up to the grade-4 cancers, in which undifferentiation or dedifferentiation ranges from 75 percent to practically 100 percent.

Broders has emphasized the fact that cancer cells arise from the regenerative layers of the epithelium, where the cells still have the capacity to reproduce, or regenerate.[6] Obviously, if a cell has become so mature or specialized that it cannot reproduce, it cannot produce tumor cells or any other type of cell. It seems logical that in the retina, as elsewhere in the body, we may expect tumors to arise from normal cells which still have the ability to reproduce, or regenerate.

Which cells in the retina dedifferentiate and give rise to tumors? In the retina, as in other nervous tissue, it is generally assumed that the highly specialized nerve or ganglion cells are completely formed at birth and never reproduce, whereas the less highly specialized glial or supporting cells retain the power of reproduction.[1, 43] As a rule, cells of least-specialized function retain to the greatest extent the simple function of reproduction, and Samuel has pointed out that it is the less highly specialized cells that most easily assume neoplastic traits.[42] Glial proliferation often has been demonstrated in benign inflammatory and degenerative retinal lesions.[1, 43] In the brain, the proliferation of astrocytes by direct division and at times by mitosis has been demonstrated by Nissl.[35] In glial tumors of the optic nerve, a gradual change from normal astrocytes to tumor cells has been observed by Verhoeff.[49] One may conclude that the most probable origin of retinal tumors is in the glial or supporting cells, either the astrocytes ("spider cells") or the fibers of Müller, or both.

The cell bodies of the fibers of Müller lie in the inner-nuclear layer, and, according to Ramón-Cajal,[40] astrocytes are present in the ganglion-cell and nerve-fiber layers: Krückmann in 1917 reported finding numerous glial cells also in the inner-nuclear and even in the outer layers of the retina. Retinal tumors arise most often in the inner-nuclear layer, frequently also in the ganglion-cell and nerve-fiber layers, in all of which glial cells are numerous, and rarely if ever in the outer-nuclear layer, where glial cells have not often been demonstrated. It is our belief that retinal tumors arise by a process of dedifferentiation or anaplasia from previously apparently normal retinal cells, usually if not always glial cells. This is in accordance with the original proposition of Virchow that tumors of the retina arise from its glial cells.[50]

The predominating cells in most retinal tumors are extremely undifferentiated and no longer have the form of glial cells. They resemble in appearance the undifferentiated cells seen in medulloblastomas of the central nervous system,[4] and in the highly malignant tumors of the adrenal medulla known as "neuroblastomas" or "sympathicoblastomas." Grinker likened these cells in the retinal tumors to the "retinoblast," a hypothetical indifferent bipotential cell produced by the primitive retinal epithelium.[21, 22] The existence of such a cell as a "retinoblast" in normal embryonic development has not been demonstrated,[4, 22] and it is a question whether these highly anaplastic tumor cells are as well differentiated as any normally developing embryonic cell. Tumors composed chiefly of such undifferentiated cells, the type frequently called rentinoblastoma, would be graded 4 according to Broders's method of classification.

Although the cells most commonly seen in retinal tumors are extremely anaplastic, at times evidences of some degree of differentiation are found. As has been men-

tioned previously, the formation of rosettes in the tumors of neuro-epithelioma type appears to be the result of partial differentiation of tumor cells, and their form is suggestive of an imitation of the primitive neural tube. Tumors with these evidences of differentiation would, in general, be classed as of grade-3 malignancy according to Broders's method; a few of the most differentiated ones might fall in grade 2.[46]

Pseudorosettes, which were numerous in practically all the tumors in which rosettes were present, and in several tumors without rosette formation, in many ways resembled the rosettes in structure and approached them in degree of differentiation. The formation of cell fibrils in the cytoplasm was also evidence of partial differentiation of these cells. Tumors with well-formed pseudorosettes appeared to be practically as well differentiated as those with true rosettes, and clinically were found to be very similar.

Tumors of astrocytoma type, such as that described by McLean, are composed of relatively well-differentiated cells and would be graded 1 in the Broders classification.

The tendency of tumor cells to differentiate to a greater or less degree explains the occurrence of well-differentiated cells, which have been reported at times in small numbers in these tumors. Spongioblasts, astroblasts, and even astrocytes have been reported by Marchesani,[32] Grinker,[21] Munoz Urra,[34] and others. Greeff[20] and others have reported in addition the presence of ganglion or nerve cells. While it is conceded that the extremely undifferentiated cells of these tumors probably have the potentiality of developing in the direction of either the spongioblastic or the neuroblastic series, it is unlikely that such highly differentiated cells as ganglion cells would be found in these highly malignant, undifferentiated tumors. The general opinion is that the ganglion cells occasionally described in retinal tumors are either normal ganglion cells that have become surrounded by tumor cells, or large undifferentiated astrocytes that have the appearance of ganglion cells when stained by nonspecific methods. Neuroblasts have never been demonstrated in retinal tumors by modern impregnation methods, according to Grinker,[22] although Bailey and Cushing[4] reported that cells of the neuroblastic series have been demonstrated at times in the corresponding "medulloblastomas" of the central nervous system. In a few tumors in our series, well-formed ganglion cells were seen, but they were in

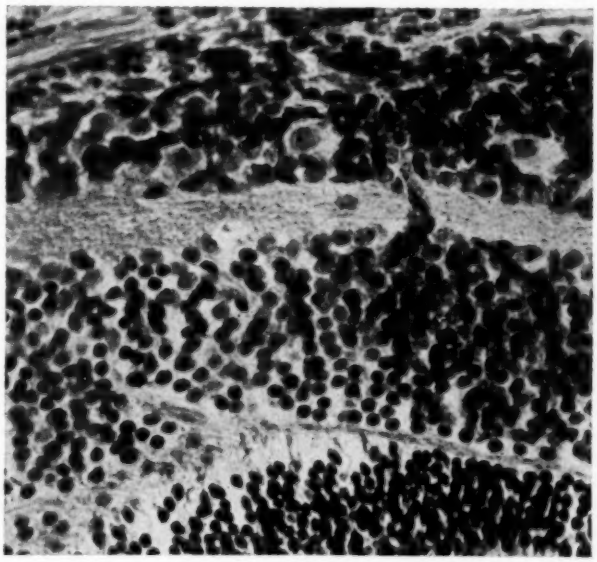

Fig. 12 (Parkhill and Benedict). Case 26. Tumor cells apparently arising *in situ* in the ganglion-cell layer and surrounding the ganglion cells which have persisted unchanged. There is malignant change in the inner-nuclear layer also (×360).

a position corresponding to the preëxisting ganglion-cell layer, and gave the appearance of normal ganglion cells surrounded by tumor cells (fig. 12).

Objections have grown to the application of the term "glioma" to the retinal tumors on the ground that they are much more malignant than are certain gliomas of the brain composed chiefly of well-differentiated glial cells; and "retinoblastoma" has been proposed as a more appropriate term.[48] Gliomas of the brain, according to the criteria of Golgi[3,19] in 1875, included only tumors composed of well-differentiated astrocytes; however, to most modern pathologists and neurologists, gliomas include all brain tumors of neuro-ectodermal or neuroglial origin, from the well-differentiated astrocytomas to the highly undifferentiated spongioblastomas and medulloblastomas. In this sense, the term "glioma" applied to the retinal tumors is exactly analogous, and includes the highly malignant tumors called retinoblastomas and those with rosette formation, called neuro-epitheliomas, as well as the extremely rare well-differentiated tumors of astrocytoma type.

TABLE 2

RESULTS IN GLIOMAS OF NEURO-EPITHELIOMA TYPE AND OF RETINOBLASTOMA TYPE (35 CASES)

	Neuro-epithelioma Type	Retino-blastoma Type
Number of cases	17	18
Cases traced	16	16
Living, no recurrence	9	2
Died of other cause	1	0
Total good results	10 (62.5%)	2 (12.5%)
With recurrence	0	1
Died of glioma	6	13
Total poor results	6 (37.5%)	14 (87.5%)

As the gliomas of the brain are divided into various subgroups according to the predominating cell type (that is, according to the degree of differentiation of the tumor cells), so may the tumors of the retina for practical purposes be subdivided according to the degree of differentiation present: (1) glioma retinae, retinoblastoma type (highly undifferentiated tumors resembling the so-called neuroblastomas or medulloblastomas of the central nervous system); (2) glioma retinae, neuro-epithelioma type (tumors showing slight or partial differentiation as indicated by formation of rosettes or pseudorosettes, with cells resembling primitive spongioblasts), and (3) glioma retinae, astrocytoma type (well-differentiated tumors, rare in the retina, made up primarily of cells nearly as well-differentiated as normal astrocytes). If other tumor types are found in the retina, they may be added to the series with a designation corresponding to the embryonic cell type that the predominating tumor cells most closely resemble; for example, glioma retinae, neuroblastoma type, or glioma retinae, astroblastoma type.

Gliomas of the retina have been regarded generally as highly malignant and with a grave prognosis. In general this is true, but, among the retinal tumors, it would appear that a group can be distinguished, composed of tumors of neuro-epithelioma type, which, if they have not invaded the optic nerve or extraocular tissues at the time of enucleation, in general have a favorable prognosis. They appear to be slower growing than the retinoblastoma type, and less often show invasion of the optic nerve or orbit. In tumors of retinoblastoma type, on the other hand, the mortality is high; and in our series, only 2 of 16 patients traced survived without recurrence for more than one year (table 2). It is noteworthy that in a large proportion of the tumors of this type, in-

vasion of either the optic nerve or extraocular tissues, or both, was present at the time of enucleation of the eye.

Summary and Conclusions

A series of 35 tumors of the retina was studied and, on a histologic basis of differentiation of tumor cells, was divided into two groups, an extremely undifferentiated retinoblastoma type, and a partially differentiated neuro-epithelioma type. A third, well-differentiated astrocytoma type, an example of which was reported by McLean, was not represented in this series. The following conclusions were drawn:

1. Neuro-epitheliomas seldom, probably never, arise from the rod-and-cone cells of the retina.

2. Gliomas of the retina of any type arise most often in the inner-nuclear layer, less frequently in the ganglion-cell and nerve-fiber layers, and rarely, if ever, in the outer-nuclear layer.

3. In cases in which regions of normal retina still persist, an appearance of multicentric origin of tumors is frequent.

4. Retinal tumors probably do not arise from embryonic rests nor from persistent primitive epithelium, but from normal cells that have dedifferentiated to produce tumor cells.

5. The cells from which the tumors arise are probably most frequently, if not always, glial cells.

6. The most undifferentiated of the tumor cells are the small cells with hyperchromatic polygonal or wedge-shaped nuclei that predominate in the retinoblastomas; they are extremely anaplastic and do not correspond necessarily to any known cell of normal retinal embryogenesis.

7. Cells in rosettes are not rod-and-cone cells and are much less differentiated than are rod-and-cone cells.

8. The formation of rosettes is an evidence of partial differentiation of the tumor cells to a point at which they resemble more or less closely the cells of the primitive neural tube.

9. The formation of pseudorosettes also signifies differentiation of the tumor cells, probably almost to the degree attained by the cells of true rosettes. The alignment of epitheliallike cells into rows and bands, sometimes surrounding blood vessels, also is a manifestation of a slight degree of differentiation.

10. Results in the retinoblastoma group were poor (only two cases with good results, or 12.5 percent of 16 traced cases), as compared with those in the neuroepithelioma group (10 cases with good results, or 62.5 percent of 16 cases traced).

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Discussion

Dr. T. L. Terry (Boston): In your work on these intraocular neoplasms have you studied specimens of so-called adenoma of the ciliary process?

Dr. Parkhill: There were none of those in our series, and I have not had an opportunity to study that type of tumor.

Dr. Terry: The reason for asking this question is because I am interested in how you might classify this neoplasm.

Dr. Parkhill: I do not believe I could answer that without examining the tumor microscopically, because our classification depends upon the histologic appearance and differentiation of the cells. It would probably be classified as an adenoma, or, if evidences of dedifferentiation were present, as a low-grade adenocarcinoma.

CEREBRAL SYMPTOMS ACCOMPANIED BY CHOKED OPTIC DISCS IN TYPES OF BLOOD DYSCRASIA

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Papilledema of the type usually designated as "choked disc" is observed at times among patients suffering from various types of blood dyscrasia in the absence of unrelated intracranial pathologic processes or involvement. The pathogenesis of this particular type of "choked disc" is of considerable interest. Usually, it has been assumed that edema of the optic disc develops in these cases as a result of an increase in intracranial pressure secondary to bleeding into some part of the brain or its meninges. Recently, we have been led to suspect the presence of an intracranial pathologic process in several patients who had blood dyscrasia, because choked optic discs were found on ophthalmoscopic examination of the patients. Since only a few instances of this type of condition have been reported previously, it has seemed worth while to discuss our observations, particularly with reference to the possible pathogenetic factors involved in the production of the edema of the optic discs.

Three of our five patients presented the picture of thrombocytopenic purpura. This disease is characterized by hemorrhagic phenomena and by a marked reduction of platelets in the circulating blood. The condition may be acute or chronic and usually affects children and young adult persons, although patients may be of any age. One of the most characteristic phases of purpura haemorrhagica is the occurrence of cyclic periods of hemorrhage and spontaneous remission. During the phase of remission the patient has complete freedom from symptoms. Frequently, the hemorrhage in such periods is of a minor nature, evidenced by only a few petechiae, so that hemorrhage may escape notice. A carefully taken history, however, will reveal the occurrence of such hemorrhage. As time goes on, these periods of hemorrhage become more frequent and hemorrhage may become more severe, so that at some point it may be decided that the acute disease itself is present, when, in reality, it is an acute exacerbation of the disease that is present. Since in aplastic anemia and acute leukemia, particularly in the leukopenic form, laboratory findings are noted that are similar to those of purpura haemorrhagica, the cyclic tendency is a valuable aid to the physician in the making of a correct diagnosis, inasmuch as this particular tendency does not occur in aplastic anemia nor in acute leukemia. Another point of diagnostic significance is the fact that hemorrhage is the initial sign and is not preceded by anemia. The significant laboratory findings in purpura haemorrhagica are: reduction of platelets, prolonged bleeding time, delayed retraction of clot, and a coagulation time of venous blood that usually is within normal limits. Ex-

amination of blood smears reveals a marked decrease in blood platelets, and there is an increase in the degree of polychromatophilia and anisocytosis attributable to demands on the bone marrow after loss of blood. Rarely, if ever, is immaturity of the myeloid leukocytes noted unless the hemorrhage has been so severe as to produce a leukemoid reaction.

The occurrence of hemorrhage into the central nervous system in the course of thrombocytopenic purpura has long been recognized. According to Longcope,[1] Wagner[2] in 1886 reported eight cases of hemorrhage into the brain and meninges. Hemorrhagic extravasation, large or small, single or multiple, may occur beneath the dura, in the meninges, into the ventricles, or into the substance of the brain or spinal cord. Such types of extravasation give rise to various neurologic manifestations such as mental confusion, paralyses, aphasia, hemianopsia, and convulsions. Geiger[3] stated that hemorrhage may be found in the central nervous system at necropsy in some cases in which the patients presented no neurologic signs nor symptoms during life. Alpers and Duane[4] believed that small meningeal hemorrhages can be found in most cases if they are searched for carefully.

Isolated reports of intracranial hemorrhage that occurred at varying intervals after splenectomy are contained in the literature. Elliott[5] reported a death, caused apparently by cerebral hemorrhage, 18 months after splenectomy. Giffin[6] reported a case in which cerebral hemorrhage caused death 15 days after removal of the spleen. McLean, Kreidel, and Caffey[7] reported hemorrhage into the left frontal lobe and left lateral ventricle that resulted fatally 25 days after splenectomy. Whipple[8] recorded observations, made by Singleton,[9] Starr,[10] and Peterson,[11] of intracranial hemorrhage as a late complication of splenectomy.

Hemorrhagic extravasation of varying extent into the retina is encountered rather frequently among patients suffering from thrombocytopenic purpura. Edema of the optic discs, however, has been noted (or at least recorded) in only a few instances among patients who have this condition. Papilledema was not noted by Eliason and Ferguson[12] in their report of 108 cases in which splenectomy was done for thrombocytopenic purpura, which they collected from the literature, and it was not noted in the 100 cases of Spence's[13] series which they accepted.

In 1882, Lawford[14] reported a "Case of double optic neuritis following purpura." A 12-year-old girl had had, during the summer of 1880, purpuric spots on the arms, legs, and body, and also several severe attacks of epistaxis. In March, 1881, after several months of freedom from purpura, she entered a hospital complaining of pain in the eyes and loss of vision. Vision was reduced to light perception in the right eye and to 20/40 in the left. Ophthalmoscopic examination revealed moderate swelling of the optic disc of the right eye with fine hemorrhages on and around the disc and mild swelling of the optic disc of the left eye without hemorrhage. Vision returned to normal within two months. Six months later mild residual pallor of the optic discs was present, but vision and fields were normal. Evidently, this was a case of acute optic neuritis and, as Nettleship[15] stated in his discussion, it is very doubtful that it was connected etiologically with the preceding purpura.

In 1923, Schinck[16] reported bilateral choked discs and paralysis of the lateral rectus muscle of the right eye in a case of thrombocytopenic purpura. He thought that these lesions were caused by intracranial hemorrhage with resultant increase in intracranial pressure. When seen first, the patient, a woman 20 years old,

complained only of diplopia and excessive bleeding. Vision was 5/5 in each eye and examination of the fields of vision revealed only enlarged blind spots. Ophthalmoscopic examination revealed bilateral choked discs of 3 diopters' elevation, with regions of hemorrhagic extravasation on the discs. Spinal puncture, performed a month later, released clear fluid that was under increased pressure. However, blood appeared in the fluid toward the end of the drainage procedure. Three days after lumbar puncture, the patient complained of vertigo, headache, and vomiting. On the tenth day after puncture choking of the discs was increased to between 4 and 5 diopters in the right eye, and to between 3 and 4 diopters in the left. Treatment consisted of intensive roentgen therapy to the spleen. Within six weeks, the lateral rectus functioned normally and choking of the discs had subsided almost completely. Within six months, the eyes were entirely normal.

In 1927, Herzfeld and Rohrschneider[17] reported loss of vision accompanied by headache, mental confusion, semicoma, and transitory diminution in hearing in a man 48 years old suffering from thrombocytopenic purpura. Ophthalmoscopic examination revealed bilateral choked discs, with elevation of 5 diopters in the right eye and 4 diopters in the left, and hemorrhagic extravasation on the discs and in the peripapillary region of the retinas. At the end of six months both optic discs were found to be pale and to have blurred margins. Vision, which at first had been reduced to such extent that the patient could count fingers only at 4 meters, had improved to 5/30 in each eye. Central scotomas could not be demonstrated. Herzfeld and Rohrschneider thought that this syndrome was caused by hemorrhage into the meninges and into the optic pathways. They did not examine the spinal fluid of the patient.

In 1933, Alpers and Duane reported two cases of thrombocytopenic purpura in which choked discs were observed. The first patient, a man 47 years old, presented slight mental confusion, motor aphasia, headache, and bilateral choked discs, elevated 3 diopters in the right eye and 4 diopters in the left, with hemorrhagic extravasation in the peripapillary part of the retina. Vision was normal. Spinal puncture revealed a clear fluid that was under a pressure of 35 cm. of water. The optic discs of the second patient, a girl four years old, were definitely choked, but it was impossible to measure the elevation. Spinal puncture revealed a yellow fluid that was under a pressure of 95 cm. of water and contained 648 erythrocytes per cubic millimeter.

In 1937, Ford[18] reported observation of bilateral choked discs in a boy five weeks old who was suffering from thrombocytopenic purpura. Necropsy revealed hemorrhage into the subdural space and into the ventricles, with hydrocephalus. Ford stated also that bleeding confined to the subdural space might cause papilledema.

To the literature as briefly referred to herein, we wish to add three cases of thrombocytopenic purpura in which bilateral choked optic discs were present without evidence of demonstrable extraneous intracranial injury, and one case (case 4) of choked optic discs in the presence of recurrent severe hemorrhage from a gastric ulcer, but without evidence of a pathologic intracranial lesion at necropsy.

Report of cases

Case 1. A woman, 21 years old, was admitted to the Mayo Clinic on July 24, 1937, complaining of uncontrolled bleeding from the nose, throat, and vagina. The tendency to bleed excessively had developed about 18 months before the pa-

tient came to the Clinic, after an attack of soreness of the mouth and throat, with bleeding of the gums. The condition had been diagnosed as being "trench mouth." The symptoms had gradually increased in severity.

At the time of this patient's admission, numerous petechiae and patches of ecchymosis were present in the skin. Blood was oozing from both sides of the nasal septum and from the vagina. A clot was present in each tonsillar fossa (tonsillectomy had been performed three weeks previous to the time of our examination). The platelet count was 30,000 per cubic millimeter. The erythrocyte count was 2,490,000 per cubic millimeter. The bleeding time was 20 minutes. The coagulation time was two minutes. There was no clot retraction. The blood smear was characteristic of the smear obtained in the presence of thrombocytopenic purpura.

Eight days after admission, the patient began to suffer from severe bilateral occipital headache, with some degree of stiffness of the neck, pain on movements of the neck, and blurring of vision. Ophthalmoscopic examination revealed extensive hemorrhagic extravasation into the retina around the margins of the optic discs, with measurable elevation of the discs (2 diopters in the right eye and between 3 and 4 diopters in the left). Hemorrhages were noted also in the periphery of the retina of the right eye. It was thought probable that bleeding into the subarachnoid space had taken place, an occurrence that would have explained both the meningeal irritation type of headache and the extensive extravasation of blood into the retina. Three days later the patient complained of severe frontal headache and increased stiffness of the neck. More bleeding had occurred into the retina.

In spite of the fact that this patient received transfused blood 18 times and a short course of radium therapy to the spleen, the platelet count did not increase to more than 70,000 per cubic millimeter and the erythrocytes decreased gradually to 990,000 per cubic millimeter. Bleeding continued and the patient died 23 days after admission. At necropsy the brain was not examined; however, cisternal puncture revealed clear spinal fluid.

Case 2. A girl, 14 years old, was admitted to the Clinic on May 28, 1939, complaining of recurrent severe epistaxis and also excessive menstruation, which had been present during the three years prior to her examination at the Clinic. A tendency to bruise easily had been noted by the patient for a year before the onset of other symptoms. Bleeding from the gastrointestinal tract had occurred at times. Transfusions of blood and the administration of moccasin venom, and roentgen rays to the spleen had not controlled the patient's bleeding tendency.

The patient was definitely anemic in appearance, and patches of ecchymosis were noted in the skin and in the mucous membrane of the mouth. The spleen was not palpable. The content of hemoglobin was 6.8 gm. per 100 c.c. The erythrocytes numbered 3,550,000 per cubic millimeter and the leukocytes 5,700 per cubic millimeter. The platelet count was 65,000 per cubic millimeter. The bleeding time was 40 minutes, the coagulation time was nine and a half minutes, and there was no clot retraction. A diagnosis of thrombocytopenic purpura was made.

On two occasions (two years, and again, several months) before examination, the patient had noted transient numbness and slight weakness of the right arm, accompanied on the second occasion by numbness of the mouth and tongue. The patient complained also of headache, which had been noted first six weeks before her admission to the Clinic. Episodes

of headache had increased in severity during two weeks, had been accompanied at that particular time by vomiting, and, one week later, by soreness and stiffness of the neck. Headache and stiffness of the neck had decreased considerably in intensity during the week previous to the patient's admission, and vomiting had been less frequent. However, diplopia had appeared 10 days before her admission. Examination of the nervous system revealed that Kernig's sign and Brudzinski's sign could be elicited bilaterally, and that paresis of the left lateral rectus muscle, with homonymous diplopia, was present. Vision was reduced to an ability to read 14/42 with the right eye and 14/112 with the left. Ophthalmoscopic examination revealed bilateral choked discs, 5 diopters in measurement in the right eye and 4 diopters in the left, with rather extensive hemorrhages into the retina, some of them definitely purpuric in type. In each macular region a hemorrhagic lesion was present, apparently in the choroid rather than in the retina. It was questionable whether the cerebral manifestations could be attributed entirely to purpura or whether they represented an independent lesion.

Splenectomy was performed as an emergency measure to control bleeding. The spleen was found to be twice the normal size, and an accessory spleen, 1.5 cm. in diameter, was found embedded in the peritoneum near the upper pole of the left kidney. Histologically, the spleen was found to be affected by chronic splenitis.

The patient made an uneventful recovery. The platelet count increased gradually, until on the twelfth day it was 246,000 per cubic millimeter. Menstruation began on the fourth day after operation and the flow was normal in quantity.

Approximately a month after her admission to the Clinic, the patient was free of headache, nausea, and diplopia. Examination of the nervous system revealed nothing abnormal. Distance vision was 6/20 and reading vision 14/25 in each eye. Perimetric fields were normal. Bilateral choked discs of 3 diopters in measurement were still present. Dr. J. Grafton Love of the Section on Neurosurgery saw the patient in consultation and advised performance of diagnostic spinal puncture and the making of an encephalogram. Dr. Love inserted a spinal-puncture needle into the third lumbar interspace, with the patient in the upright position. The column of cerebrospinal fluid rose in the manometer to 47 cm., a height equal to the vertex of the head. Ninety cubic centimeters of fluid was removed fractionally and was replaced by 80 c.c. of air. The encephalograms were excellent and showed normal filling of the ventricular system without evidence of shift. A small quantity of air was present in the subarchnoid space. There was no evidence of an expanding intracranial lesion.

The spinal fluid was clear and contained 5 lymphocytes per cubic millimeter. Total protein content was 50 mg. per 100 c.c., and no globulin was present. Results of the Kolmer and Kline tests were negative.

At the time the patient was dismissed from the hospital she was essentially symptom-free, and papilledema had receded to approximately 2 diopters.

Case 3. A girl, 14 years old, was admitted to the Clinic on November 30, 1939, because of bleeding from the gums, recurrent severe epistaxis, excessive menstrual flow, and patches of subcutaneous ecchymosis. The bleeding tendency had been noted first a month prior to the patient's admission to the Clinic. For three days prior to her admission, the patient had suffered from episodes of nausea and vomiting.

Examination confirmed the presence of

extensive purpuric regions over the trunk and extremities, and of oozing from the gums and nasal mucous membranes. Blood pressure was 145, systolic, and 98, diastolic, expressed in millimeters of mercury. The content of hemoglobin was 3.15 gm. per 100 c.c. of blood. Erythrocytes numbered 2,560,000 per cubic millimeter and leukocytes, 39,000 per cubic millimeter. The platelet count was 58,000 per cubic millimeter. Bleeding time was 30 minutes, coagulation time 8 minutes, and clot-retraction time 7¾ hours. The Quick prothrombin time was 19 seconds. Morphologically, the blood was characteristic of the condition of the blood in the presence of thrombocytopenic purpura, with increased regeneration of erythrocytes and occasional immaturity of myeloid leukocytes.

Because of the patient's poor general condition, splenectomy was performed 12 hours after her admission, as an emergency procedure. Three small accessory spleens also were removed. The spleen was of about twice-normal size and histologically was seen to have evidence of chronic splenitis with hyperplasia of the follicles and endothelial proliferation of the sinuses. Megakaryocytes were not seen.

The patient had a stormy convalescence. Intercurrent pulmonary and renal infection developed. On the eighth day, however, the bleeding time had decreased to three minutes; by the fourteenth day the platelet count had increased to 164,000 per cubic millimeter, and the patient displayed no further evidence of bleeding diathesis during the remainder of her stay in the hospital. On the third day after her admission, the patient complained of diplopia, and paralysis of both lateral rectus muscles was noted. On the tenth day after her admission, ophthalmoscopic examination revealed bilateral papilledema between 3 and 4 diopters in measurement in the right eye and between 2 and 3 diopters in measurement in the left eye. A few regions of hemorrhagic extravasation were present in each retina, near the optic discs. Reading vision was 14/21 in each eye and the visual fields were normal to the confrontation test. There were no other gross evidences of the presence of a lesion of the central nervous system. At the end of two and a half months, papilledema had completely subsided and the hemorrhages had essentially disappeared from the retina. The excursions of the lateral-rectus muscles were essentially normal. When the patient returned to the Clinic for observation six months later, vision, visual fields, ocular muscles, and ocular fundi were entirely normal.

Case 4. This case is not one of thrombocytopenic purpura. A man, 50 years old, was admitted to the Clinic on January 30, 1937, for treatment of recurrent bleeding from the stomach. He stated that he had had what he called a "sour stomach" all his life, and that for six years he had experienced pain through the region of the heart when his stomach was empty. Three years before admission he had had a hemorrhage from the stomach sufficiently extensive to require hospitalization and transfusion of blood. At that time he had noted some disturbance of vision, a disturbance which apparently had been corrected by refraction. For three months the patient had noted that when he would rise from a stooping position the vision in his left eye would be blurred for several seconds. The cause of this disturbance of vision was not clear. Two weeks before his admission gross bleeding from the stomach had occurred again.

On this patient's admission, the results of a general physical examination were not remarkable except for generalized

abdominal tenderness. Blood pressure was 146, systolic, and 98, diastolic, expressed in millimeters of mercury. The content of hemoglobin was 13.1 gm. per 100 c.c. of blood. The erythrocytes numbered 3,640,000 per cubic millimeter and leukocytes, 7,500 per cubic millimeter. Serologic tests of the blood for syphilis revealed positive reactions to the Kline, Kahn, Hinton, and Kolmer procedures. A clinical diagnosis of syphilis could not be established, however. Roentgenologic examination of the stomach revealed a large penetrating ulcer situated fairly high on the lesser curvature of that organ.

With the proper refractive correction, vision was 6/4 with the right eye and 6/6 with the left eye. The fields of vision on the perimeter and on the tangent screen at 1 meter were normal, except for an enlargement of the left physiologic blind spot. Ophthalmoscopic examination revealed in both eyes sclerosis of grade 1 of the retinal arterioles and engorgement of the retinal veins, and in the left eye a choked disc 3 diopters in elevation was noted. Roentgenograms of the head disclosed nothing abnormal nor did examination of the nervous system.

When the patient was placed in the hospital, oozing from the gastric ulcer continued, and on the eleventh day another gross hemorrhage occurred; the hemoglobin content was reduced to 6.6 gm. per 100 c.c. and the erythrocyte count fell to 1,380,000 per cubic millimeter. Five days previously the patient had fainted in the bathroom and stated that he had struck his head, although there were no external signs of injury. At this time he complained of severe headache. Ophthalmoscopic examination revealed an increase in the elevation of the choked disc in the left eye to between 4 and 5 diopters and a choked disk of 1 diopter's elevation in the right eye. No positive neurologic signs were present, and a second roentgenogram of the head disclosed nothing abnormal. Spinal puncture in the fourth lumbar interspace revealed a clear fluid that was under 40 cm. of water pressure. The fluid contained only 1 lymphocyte per cubic millimeter and had a total protein content of 20 mg. per 100 c.c. Negative results were obtained from the test for globulin, and from the Nonne, Kahn, and Kline tests. The colloidal gold curve was normal. Headache was relieved after drainage of the spinal fluid.

In spite of repeated transfusions of blood and surgical excision of the gastric ulcer, the patient died on the eighteenth day after admission. At necropsy no pathologic changes were found in the brain nor in the meninges. An antemortem thrombus was found in the right cavernous sinus with some extension into the left. However, the pathologist considered this thrombus to be of less than 72 hours' duration.

Comment

The mechanism responsible for the origin of the choked discs in these four cases is not clear. In case 1, it is possible that the apparent edema of the disc and retina was a local reaction to the extensive bleeding into those tissues. The episodes of severe headache and stiff neck suggest also that recurrent hemorrhage into the subarachnoid space might have occurred. However, spinal fluid obtained at necropsy showed no evidence of old nor of recent blood. In case 2, also, the spinal fluid was found to be clear, and no positive evidence of a space-occupying intracranial lesion could be obtained. In case 3, aside from the choked discs and paralysis of the lateral rectus muscles, there were no signs of a gross cerebral lesion. In both case 2 and case 3, the regions of hemorrhagic extravasation into the retina

at the time of observation of the papilledema were not more extensive than might be expected in association with the average choked disc as encountered in cases of cerebral neoplasm. Choked discs are encountered rarely in association with intracerebral hemorrhage, a condition which usually is rapidly fatal to the patient. Choked discs develop rather late in the course of subdural bleeding or hematoma, and the papilledema and other symptoms present in such cases rarely subside until the hematoma has been drained. In cases of spontaneous or posttraumatic bleeding into the subarachnoid space, choked disc usually is a relatively late sequel to the venous obstruction that results from the presence of blood in the subarchnoid spaces of the optic-nerve sheath, and appears, as a rule, at least several days after the initial occurrence of multiple hemorrhages into the retina. It would seem logical, therefore, to assume that some factor other than actual intracranial bleeding might be responsible for the development of the papilledema in these cases.

In case 4, since no general hemorrhagic tendency was demonstrated, it did not seem logical to assume that the edema of the optic discs was secondary to intracranial bleeding, and no evidence of cerebral hemorrhage was found at necropsy. In the presence of normal spinal fluid it would not seem logical to consider the papilledema in this case to be of syphilitic origin, in spite of the positive results obtained in serologic study of the blood. It would seem most probable that the edema of the optic discs observed in this case was associated in some way with the loss of blood. Whether the headache and the increase in pressure of the spinal fluid observed on one occasion were an expression of edema of the brain resulting similarly from loss of blood or whether they resulted from the presumed injury to the head, it is impossible to say.

The only factor common to all these four cases that might be assumed to be responsible for the development of choked optic discs was loss of blood. It is rather generally recognized that loss of vision, sometimes permanent, may result from sudden severe loss of blood. Various theories have been advanced to account for loss of vision such as was described herein. These theories, which have been summarized by Groenouw,[19] include postulation of the occurrence of hemorrhage into the sheath of the optic nerve, lymph-edema in the sheath of the optic nerve as a result of edema of the brain and consequent filling of the meningeal spaces with lymph, edema of the visual centers, minimal air embolism, and lowered nutrition of the retina and optic nerve, caused by low blood pressure. In the light of present knowledge of the dependence of the retina and optic nerve on an adequate supply of oxygen and of the rapid and permanent loss of function that results from short periods of deprivation of oxygen (as in spastic closure of the nutrient arteries), it would seem logical to assign the cause of the loss of vision in these particular cases to anoxemia. It is probable, also, that the edema of the discs and retina that develops in the presence of so-called angiospastic retinitis is caused by anoxemia. It seems possible, therefore, that the reduced oxygen-carrying power of the blood resulting from acute or chronic loss of blood may lead to the production of anoxemia of the tissues of the optic nerve and retina sufficient to induce visible papilledema. Certainly it is true that in many cases of severe anemia resulting from any cause there is a suggestion of edema, ophthalmoscopically, in the optic nerve and retina. Experimentally in animals, Holden, as quoted by Groenouw, found edema of the nerve-fiber and

ganglion-cell layers of the retina with early degeneration of the ganglion cells 12 days after loss of blood. Four weeks after loss of blood he observed progressive degeneration of the ganglion cells and of the medullary sheaths of the optic nerves. In his discussion of the changes observed in the optic discs in the presence of the hemorrhagic diatheses, Schinck[16] stated that Seggel had reported the occurrence of bilateral choked discs of 4 to 5 diopters' elevation in a case of scurvy, that Hirschberg had reported edema of the discs with small regions of hemorrhage on the discs and in the retina in a case of hemophilia, and that Hessberg had reported bilateral choked discs and inferior altitudinal anopsia eight days after severe hemorrhage in a case of hemophilia. One of us (Wagener) has observed bilateral choked disc of 3 diopters' elevation (without hemorrhage) and paresis of the lateral rectus muscle of the left eye in a patient who had hemolytic icterus and severe secondary anemia. Encephalographic studies failed to reveal the presence of an intracranial lesion. The patient, a boy five years old, improved rapidly after splenectomy and the choked discs receded gradually without additional treatment. All these observations suggest that edema of the optic discs can develop as a reaction of the tissues to local deprivation of oxygen. It is possible that edema of the brain may develop in these cases for a similar reason and that such edema may be responsible, in part at least, for the edema of the optic discs and paralysis of the lateral-rectus muscles observed in our cases. It is a well-known fact that choked discs and paralysis of the extraocular muscles may occur as results of that type of edema of the brain that is associated with cerebral concussion without evidence of other types of intracranial injury.

Summary and Conclusions

Three cases of thrombocytopenic purpura are reported in which bilateral choked optic discs were present in the absence of a demonstrable extraneous intracranial pathologic process or lesion. In none of these cases could it be proved that intrameningeal or intracerebral bleeding had occurred.

Another case (case 4) is reported in which choked optic discs appeared in the course of recurrent severe hemorrhage from a gastric ulcer. In this case an intracranial pathologic process or lesion could not be demonstrated at necropsy. It is suggested that the mechanism of production of papilledema was essentially the same in these four cases: a local reaction of the tissues of the optic nerves to anoxemia that resulted from loss of blood.

It is concluded that edema of the brain arising from the same cause may be a factor in the development of the papilledema and of the paralysis of the lateral-rectus muscles at times associated in such cases.

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FORMALINIZED HETEROGENEOUS AND HOMOGENEOUS CORNEAL TRANSPLANTATION—EXPERIMENTAL*

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One of the most difficult problems in corneal transplantation is the procurement of adequate material which can be used as "grafting" tissue. Unless one is fortunate enough to do keratoplasties with great regularity, it is not always possible to have a "fresh" cornea available at the right moment. This, in addition to the fact that it has been reported in the European literature that fixed corneal tissue has been used with great success in transplants, both experimentally and on human beings, brought to our attention the need for work of this sort.

Our idea of using fixed tissue as material for keratoplasty was inspired by the work of Salzer,[1] who first reported the use of formalinized corneae in transplantation on rabbits; however, these were only of partial thickness and, therefore, cannot be classed as truly successful transplants. In 1912, Magitôt[2] reported the use of cornea preserved in "Locke's solution," then transferred to hemolyzed blood serum and kept at 5°C. He was able to keep these tissues active for as long as one week; the graft he refers to came from an eye which had had a marked glaucoma. This, too, was only a "partial" graft going only to Descemet's membrane. Incidentally, at this early time, Magitot referred to the use of the cornea from still-born infants as a possible source of material.

In 1926 Koppanyi and Byfield[3] used the mesentery in a corneal transplant on a rabbit, and with apparent success. However, from the description, it is difficult to ascertain whether this was a through-and-through corneal graft or one of only partial depth; the graft did not become transparent.

Most of the work since 1911 has been done with the idea of confirming or disproving Salzer's original concept. It was his belief that the graft acted only as a guiding structure for the ingrowth of new tissue from the cornea of the recipient. In other words, it is not a matter of transplantation, but one of replacement or regeneration. The following is noted by MacCallum[4]: "It is important to observe that in some cases, as in transplantation of bones or nerves, the graft may survive only as a sort of splint which supports and guides the new growth of tissue from the host, which finally absorbs and replaces it entirely."

In recent years, many men have used cold as a method of preserving cornea; this has been advocated chiefly by Filatov[5] and used also by Castroviejo.[6] They use temperatures of 2° to 3°C. as the agent, keeping the eye in Ringer's solution, or normal saline. This method slows the process of autolysis and is borne out by the report of Filatov and Bajenova,[7] who preserved cornea at −2°C. to verify the vitality of dried cornea of the rabbit by tissue culture; they found that with a loss of 76.3 percent of its initial weight, the cornea is capable of growing, that is to say, "preserves its vitality." Cornea preserved in this way must be used in a comparatively short time. Fine[8] after some experimental work on cornea preserved in citrated blood at 4°C., concluded that fresh material, or that kept for only 24 hours, afforded better results than if kept for 48 hours or more.

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Thomas,[9] trying heterogeneous corneal transplants in rabbits, used grafts from one human and five cat corneae. He concluded that heterogeneous grafts can unite, but unlike successful homogeneous grafts, they do not remain transparent and become vascularized. Tissue from the recipient eye grows in and tends to envelop the graft. He[10] also used scleral tissue as material for tranplantation and found that it could be used to replace the whole thickness of cornea. After a rather long period of time (two years or so) they developed an epithelium and endothelium derived from the tissues of the recipient eye. The replacement of the graft tissue by this new tissue is accompanied by a considerable degree of transparency in the graft. It was also found that those which developed greater vascularity tended to become less opaque, and Thomas thought that the vascularity had a stimulating effect on the ingrowth of new corneal fibers. This postulate is open to question, for some believe that vascularity is a precursor of connective tissue and opacity.

Petrosiantz,[11] in a study of biomicroscopy of what he calls "conserved cadaver eyes," used the slitlamp to observe daily corneal changes of eyes kept at +2° to +4°C. He found that the epithelium becomes opaque, irregular, and edematous by the seventh day; by the fifteenth day the cornea is only semitransparent, and by the twentieth to the twenty-fifth day, it is completely opaque. The clinical data following the use of this tissue prompted the conclusion that the foregoing changes in the clarity of the cornea and regularity of the epithelium have apparently no effect on the transplant. This is of interest here, for formalinized corneae are extremely opaque.

The report of Löhlein,[12] who took part in the discussion of a topic entitled "Progressive corneal transplantation" before the German Ophthalmological Society in 1938, was probably responsible for much interest in the use of formalinized cornea. He stated, "one does not expect a permanent survival of the transplant but a gradual displacement by surrounding tissue." This included a report of two cases in humans in which formalinized cornea was used; one of these was a bilateral transplant—one eye utilizing fresh cornea, the other, formalin-fixed cornea. The result with the latter was better than the one in which fresh cornea was used. A comment on this paper was made by Elschnig,[13] who agreed that it must be assumed that the implant becomes replaced by new growths of corneal tissue.

Incidentally, our work was begun before Löhlein's article was printed in this country (which was in February, 1940) since which, at least two investigators have reported on the value of formalinized corneal tissue in transplantation. Peculiarly enough, they both appeared in the same bound volume of Ophthalmologica, one work being that of a Russian, the other of a German. The former, Scherschewskaya,[14] gave a very complete and praiseworthy analysis of his experimental work. He followed the method of Weidenreich, and kept the cornea in 4-percent formaldehyde for two days, washed it in Ringer's solution for four days, then allowed it to stay in Ringer's for four more days, changing the solution daily. The surgical procedures were done using Filatov's technique. Of 37 rabbits operated upon, he reported "healing" in 12 (or 32 percent)—one glimmering, three transparent, and eight opaque corneae resulting. This technique was also used in five cases of human eyes (all of which were unfavorable to begin with because of the uncertainty of the ultimate result), and he obtained "one transparent, one glimmering, and two turbid healings." This author claims there is no greater

healing reaction to formalized cornea than to the "fresh" transplant. He also agrees with Salzer's findings, mentioned previously, and in regard to the long time that it takes for the resorption of the graft and its replacement through regeneration. Other investigators, such as Ascher, Weidenreich, and Bussaca, are quoted by Scherschewskaya as furnishing encouraging results in this line of endeavor.

Following Löhlein's work, Deutman[15] also used formalinized cornea in keratoplasties on rabbits. He kept the corneae in 4-percent formaldehyde for only 24 hours, then washed it in running water for 48 hours. In this comparatively small series of five rabbits, two were operated upon with Castroviejo's technique, and two with the Franceschetti trephine. All the transplants "took" and healed well with less reaction than when living cornea was used. After an observation period of 3 to 11 months, none of the grafts was clear, the opacities being located in the posterior layers of the corneal parenchyma. Deutman concludes that keratoplasty with formalinized cornea is more fit for tectonic purposes than for optical improvement.

Experimental

With Salzer's work as a foundation, we decided to attempt penetrating grafts of formalinized cornea. The basis of the use of fixatives in preserving tissue is that it kills living cells by stopping enzymatic processes associated with life; and also, it coagulates protein. Formalin, used here in 10-percent solution, is probably the most widely used fixative, and for that reason our experiments were begun with the use of this medium.

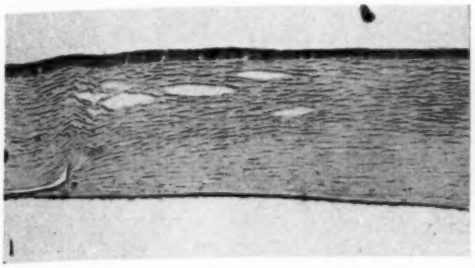
In one group of rabbits, an attempt was made to graft formalinized dog corneae. The cornea was removed from the eye of a dog that had been killed in the surgical laboratory, and the tissue immediately dropped into the formalin, where it was kept for from 7 to 28 days until the day preceding the operation. It was removed from the formalin 24 hours before the time it was to be used and washed with running tap water until a few minutes before the operation, being transferred then to sterile saline. One dog's cornea was used for 4 different grafts, a single quadrant being taken each week; in this way some idea of the optimum time of fixation might be gained. Five rabbits were operated upon, using this type of tissue; in four of these, the graft "took," and remained in place; in one, the graft was expelled following an infection. None of these grafts became clear, even over a period of 1 year's observation; there was a moderate amount of postoperative reaction, being only slightly more than that seen following rabbit-to-rabbit grafts of similar tissue; vascularization began within 7 to 10 days, reached its peak in about two weeks, and then gradually subsided. The length of time the cornea had been kept in formalin seemed to make little difference.

In the second group of animals, formalinized rabbit corneae were tried, the donor material being "fixed" for ten days to one month before being used. For the first 4 transplants in this series the technique of washing was the same as that used before; one graft of this group became infected, the other three healed well. To reduce the amount of reaction, the method of washing the cornea was changed; instead of using running tap water, we followed a more sterile technique by washing with water that had passed over a Berkefeld filter. Five rabbits were used in this group; only one mild infection occurred in which case the graft did not slough out. The postoperative reaction was definitely less than when dog corneae were used, and the

transplants washed with water passed over the Berkefeld filter exhibited less swelling and redness than the others. Of the nine grafts in this group, seven healed well; anterior synechiae developed in three cases; in one, the iris was attached to the transplant, and in the other two, the iris was adherent to the wound at the upper limbus. Three grafts in this group are definitely clearing.

slough. No transparent grafts were obtained. The postoperative reaction was essentially the same as that seen following keratoplasties in which were used formalinized corneae washed with water that had passed through a Berkefeld filter; vascularization was noted during the first and second weeks, gradually subsiding in intensity as the operative reaction quieted.

As already stated, none of the hetero-



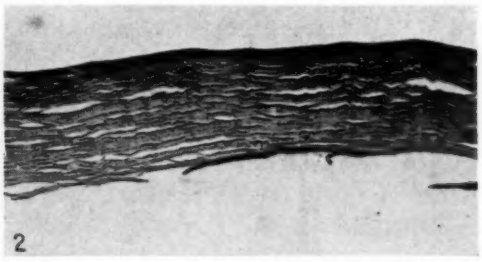


Fig. 1 (Wiener and Rosenbaum). Section of dog-to-rabbit transplant of formalinized cornea described in detail as no. 3023.

Fig. 2. Section of rabbit-to-rabbit transplant of formalinized cornea described in detail as no. 3-65.

Because of the opaqueness of the grafts, despite the fact that the "takes" were numerous, it was decided to try another fixing solution; we assumed that too much tissue damage was done by the formalin. We obtained from the research pathology laboratory some "Petrunkowitsch solution" (paranitrophenol, 60-percent alcohol, ether, cupric nitrate, nitric acid). This was used originally in the tropics because it maintained its properties for an indefinite period of time at relatively high temperatures. The corneae were placed in this solution for 12 hours, then in three separate changes of 70 percent alcohol for 30-minute periods; they were then transferred to sterile saline and kept in the ice box until used. In the third series of rabbits operated upon, corneae were used that had been kept in this solution for from 7 to 24 days. Eight rabbits comprised this group; seven grafts "took," one became infected, but did not

geneous transplants cleared, though four of the five healed. A histological report of one of these, made by Dr. Harvey Lamb, follows:

"Microscopic examination of (fig. 1, no. 3023) the dog's corneal transplant to the rabbit's cornea (1 year postoperative) presents a perfectly smooth union of the adjoining surfaces. The thickness of the transplant is a little greater than over the remainder of the cornea. This change in thickness is not abrupt but gradual. Just beyond the end of the transplant, the part of the rabbit's cornea farther from the corneoscleral junction is somewhat thicker than the corresponding part of the rabbit's cornea of the opposite side.

"The anterior epithelium of the cornea proceeds smoothly and unaffected from the surrounding rabbit's cornea over the surface of the transplant. Bowman's membrane is not present in dogs' or rabbits'

eyes. The individual lamellae of the substantia propria have joined smoothly end to end, although at one side, with the formation of a sharp angulation or wave.

"The limits of the corneal transplant are shown only by the break in Descemet's membrane. On one side, the end of Descemet's membrane belonging to the surrounding rabbit's cornea is turned anteriorly a little; on this side, a second thickness of Descemet's membrane lies peripheral for quite a distance to the transplant, just behind Descemet's membrane of the rabbit. This second posterior layer of Descemet's membrane has probably been derived from loose posterior endothelium of the dog's transplant. On the opposite side of the transplant, Descemet's membrane gradually thins out and disappears before the rabbit's cornea is reached. Just beyond the end of the transplant on this side and behind the rabbit's cornea, the absence of Descemet's membrane continues for a short distance, and the posterior endothelium is considerably degenerated here.

"In the dog's transplanted cornea, the anterior epithelium is somewhat irregular in thickness, and the epithelial cells under the superficial flattened cells of the anterior epithelium are very irregular in arrangement. Anteriorly, in the dog's transplant and in the adjacent rabbit's cornea on the side nearer the corneoscleral junction, the corneal corpuscles present a mild proliferation and the formation of a few macrophages."

A microscopic section of one of our better rabbit-to-rabbit grafts, 9 months postoperative, was also examined by Dr. Lamb and reported as follows:

"The formalinized corneal transplant (fig. 2, no. 3065) is a little thicker than the surrounding rabbit's cornea. The anterior epithelium of the surrounding rabbits' cornea is smoothly continuous with that over the transplant. The lifting of the epithelium in places over the transplant is an artifact, being also observed over the surrounding fixed part of the cornea. The thickness of the anterior epithelium is of good normal degree over the transplant.

"The lamellae of the substantia propria meet well end to end at the line of junction between the formalinized transplant and the surrounding fixed part of the cornea. The individual lamellae throughout the transplant and surrounding cornea have the same thickness and the same good staining of the corneal corpuscles. A few small capillaries are present between the anterior lamellae of the corneal transplant and the part of the surrounding cornea nearer the corneoscleral junction.

"No posterior endothelium or Descemet's membrane is present behind the transplant itself."

In our experiments the Wiener-Alvis punch was used as the instrument for the transplants. The advantages of this instrument have been brought out previously,[16] namely: 1. The segments are rapidly obtained. 2. They are identical and therefore fit exactly. 3. The beveled edge prevents the graft from slipping into the anterior chamber. 4. The lens is protected so that it cannot be injured in the procedure.

The technique[17] is almost the same as originally described with the exception of a slight modification in the corneoscleral suture and in the suture used to keep the graft in place. These changes were necessitated by the difficulty in working on the animal eye.

Operative procedure. Anesthesia is obtained by injecting Dial (Ciba), 2 c.c. per Kg. of body weight, intravenously (using a vein in the ear of the rabbit). Three or four drops of metaphen solution (1:2500) are instilled; a drop of 1:100 adrenalin is then used to dilate the

pupil. Using the corneoscleral suture as described by McLean[18] for cataract extraction, a slight groove is made in the cornea at the upper limbus; a thin, silk suture on a Kalt corneal needle is passed through the lips of the groove, entering and emerging as close to the corneal incision as possible. The suture is then drawn out of the groove with a blunt

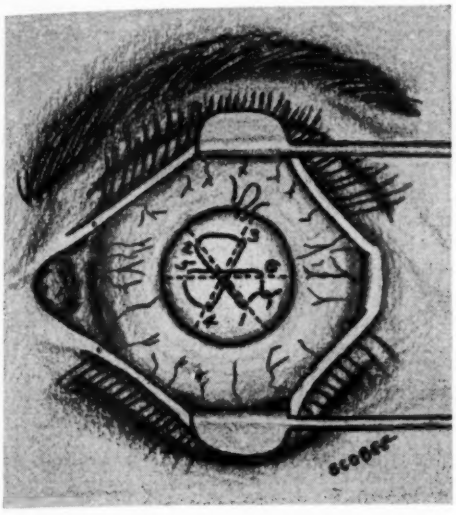


Fig. 3 (Wiener and Rosenbaum). Drawing showing the placement of sutures.

iris hook and the loops laid aside. (This differs from the original McLean suture only in the absence of a conjunctival flap, and the lack of exposure makes this omission necessary.) The center of the cornea is marked with a scalpel point, and throughout it three lines are lightly drawn with the scalpel point, dividing the corneal surface into six approximately equal parts (fig. 3). A drop of fluorescein is instilled and then washed out with saline.

With a long, single-armed suture, a superficial bite is taken in the cornea about 4 mm. below the center and to the right of the line at 1, emerging on the cross line at this point. It then is placed into the cross line at 2, about 4 mm. above the center, emerging just to the right of line; enters again just to the left at 3 and emerges on the line, etc. The last bite is taken entering on the line at 6 and coming out just below so that the suture may be tied there. The loops are then turned carefully aside.

An incision with a keratome is made in the upper limbus going through the prepared groove and enlarging the opening as the keratome is withdrawn; this may have to be further enlarged with the scissors. The female blade of the punch is now introduced into the anterior chamber, adjusted so that it will be in the center of the cross-lines, and the segment punched out. The piece of cornea comes out attached to the punch. The corneoscleral suture is immediately tied. The segment from the donor cornea is removed and gently moved into position with the scalpel point. The loops of suture are taken up and the suture tied rather loosely. Atropine is instilled and the lids are sewed together with three interrupted silk sutures.

The sutures are removed on the fourth postoperative day. For this, four or five drops of 0.5-percent pontocaine are instilled to anesthetize the cornea. Another drop of atropine is used and some xeroform powder sprinkled in the conjunctival sac.

Discussion

There were 22 keratoplasties done in this experiment, 5 of which were formalinized dog cornea to rabbit, and 17 were chemically fixed rabbit cornea to rabbit. The first group was carried out with the hope of demonstrating a form of heterogeneous graft that might be successful. These transplants did not slough out as one would expect had fresh dog's cornea been used; however, the postoperative reaction was definitely more intense and the vascularity greater than in the rabbit-to-

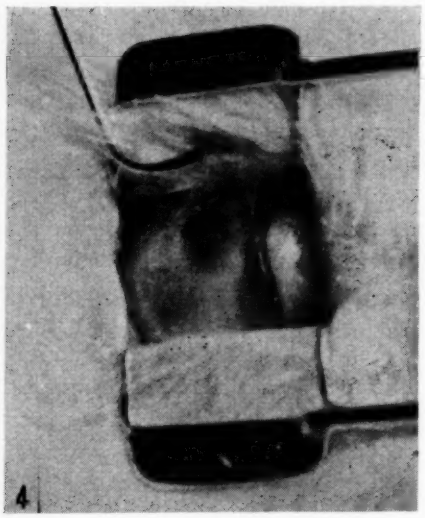


Fig. 4 (Wiener and Rosenbaum). Rabbit-to-rabbit graft of formalinized tissue 9 months postoperatively. The transplanted area was still not clear but was definitely less opaque than it had been. Figure 2 is a microscopic section of this cornea.

Fig. 5. Rabbit-to-rabbit graft 8 months postoperatively. This corneal scar was also clearing gradually but retained considerable opaqueness.

rabbit grafts (fig. 4). The specific substances which make fresh heterogeneous grafts unlikely are evidently less reactive when the tissue is fixed.

In the second group, 9 formalinized rabbit-to-rabbit transplants were attempted; of this number 8 healed. Three of these showed definite signs of clearing. The time required is long and the process a gradual one (fig. 5, 6). From the histological sections one sees an orderly replacement or ingrowth of new corneal fibers with corneal corpuscles evident. In this connection, an interesting observation is noted by Nagiotte[19] who, though he was not working with transplantation believed that the cornea may become invaded by leucocytes which are not to be distinguished from normal cells of the cornea, and that they take on the appearance, function, and activity of corneal corpuscles from which they cannot be differentiated.

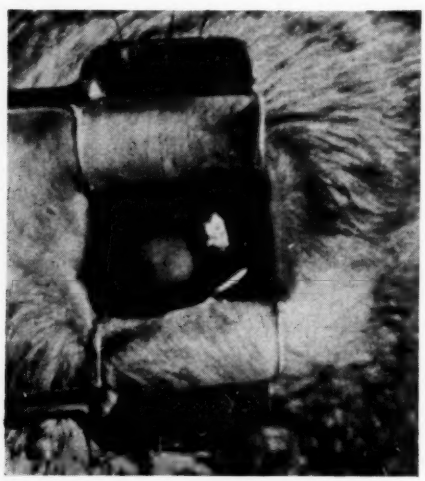


Fig. 6 (Wiener and Rosenbaum). Dog-to-rabbit graft 12 months postoperatively. It was from a similar cornea that section in figure 1 was made. These scars were definitely more opaque than rabbit-to-rabbit grafts.

It is evident from these sections that the epithelium and endothelium are derived from similar layers of the host. Descemet's membrane, on the other hand, because of its "curling" at the margins of the graft, is probably a true remnant of the donor material. The fate of the stroma of the graft and the origin of the stroma as finally seen is questionable. If

this is a true replacement it is an extremely orderly one, and yet this must be the *modus operandi*, for surely one cannot conceive that dead tissue, such as formalinized cornea undoubtedly is, could be revitalized. This question could probably be answered by histological examinations of this type of graft at graded intervals following the operation.

The results in this group seemed better than those in the others and certainly warrant further experimentation. It is more than likely that clearer grafts are obtainable with a longer period of washing.

Eight grafts were made using cornea fixed in Petrunkowitsch's solution; seven of these "took." The reaction was essentially the same as those with formalinized material; certainly it was no less. The transplants themselves seemed slightly more cloudy and did not tend to clear nearly so rapidly as some of those in group 2.

One of the most striking features of doing keratoplasties with "fixed" corneae is the ease with which the material is handled. When using fresh cornea, particularly in the rabbit, it is often difficult to ascertain which side is epithelium and which is endothelium because of its thinness and softness. With formalinized cornea, this difficulty is lessened because of the increased thickness and firmness of the tissue.

We wish to thank Mr. Alfred How for his kind and efficient assistance in this work.

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THE TOXIC SYNDROME OF OCULAR TUBERCULOSIS*

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The injection of tuberculin has an effect on the general condition of the organism, favorable or unfavorable according to the dosage employed. A new injection ought not to be made without taking into consideration the quality of this general reaction, whether to increase or diminish the strength of the solution employed, or to continue or suspend the treatment. In this exploratory investigation of the general reaction, inquiries should be made as to the appearance, the appetite, sleep, energy, weight, temperature—every departure from the normal is a signal for caution.

When making an investigation previous to the injection of tuberculin it was surprising to find with great frequency an interesting, unexpected phenomenon. All or almost all the patients who under tuberculin treatment experienced an improvement in their ocular affection likewise presented an improvement in appetite, sleep, energy, weight, and the like—and even at times a manifest change in appearance.

Thus a patient with iritis stated not only that he saw better, but, surprisingly, that he ate more, slept better, had more energy for his work, and on weighing himself before his visit, found that he had gained from 500 grams to a kilo in weight.

This unexpected phenomenon put us on guard, and as it was repeated several times, we paused to consider it attentively. Its study made us devise a new horizon, as we shall now see. If, under tuberculin treatment, there should be an improvement in the general condition, it must be that appetite, sleep, energy, and weight were not normal before the treatment.

From that time on, in the observation of every patient, after studying the ocular affection, the general condition was also inquired into, and on the respective history cards were described the pertinent data which were then sent with the patient to the tuberculosis consultant. From accumulated observations has emerged the following clinical conclusion: Tuberculin-sensitive ocular affections are accompanied by a characteristic change in the general condition, consisting of headaches, anorexia, gastrointestinal changes, insomnia, somnolence, asthenia, loss of weight; in women, dysmenorrhea—in certain cases also night sweats, fever, and an appearance of illness. In other words, tuberculin-sensitive ocular affections are accompanied as a rule by a toxic syndrome.

This toxic syndrome, with parallel development, changes with the ocular disease. If under tuberculin therapy the ocular affection improves, simultaneously there is an improvement in the general condition, and vice versa. Moreover these changes in the ocular and general condition can be induced spontaneously, without being influenced by tuberculin. Thus the outbreak of an iritis is usually preceded by loss of weight, anorexia, asthenia, and headache over a period of

* Read before the first Pan-American Congress of Ophthalmology, in Cleveland, October 11, 12, 1940.

several weeks.

A relation between the two pathological phenomena, the local and the general, seemed inescapably established, and clinically we could not refrain from accepting a priori that both were the result of a common cause. I say clinically on purpose, for the clinician speaks only of possibilities and probabilities, and never trusts his diagnosis completely. For the physician walks on the variable ground of the biological sciences, not on the rock of the mathematician, and this hesitating, perilous way has a particular attraction. Clinically we should deduce this conclusion from the facts.

What was this X factor which provoked at the same time the ocular and general affections? Cases of scrofula in the adult, that is to say, adults ill with the syndrome of infantile scrofula, who showed absolutely the same toxic syndrome as our patients with iritis, keratitis, and scleritis, answered this question and solved the pathogenic and etiologic problem.

The following data are among many observations in this regard:

Case 1. María G. B., aged 24 years, was hospitalized from the Clinic because of a bilateral blepharo-kerato-conjunctivitis on a scrofulous-eczematous basis. For a year she had suffered with her eyes, but previously had never had ocular irritation. The illness began simultaneously with edema of the face and coryza. There were facial scrofula, blepharospasm, intense bulbar injection, multiple corneal ulcers with adherent leukoma, superficial vascularization of the cornea, and chronic blepharitis. She also presented the typical toxic syndrome peculiar to glandular tuberculosis: (a) headache—frequent and at times so violent that it kept her awake at night; (b) insomnia—she was awake up to three nights in succession, insomnia alternating with somnolence; (c) somnolence—she slept day and night and had to be awakened for meals, eating and then going back to sleep; (d) anorexia—she had no desire to eat at any time, and could be urged to take only fruit, salads, tea, and maté; (e) asthenia—she walked with the body slumped and had no desire to get up; when awake she remained in a reclining position, having no desire to sit up; (f) night sweats—some nights she had cold sweats; for six successive nights the sheets were soaked, and at times had to be changed during the night; (g) dysmenorrhea; (h) emaciation—"I became very thin and my clothes became so loose the pockets looked like hen coops."

The Mantoux reaction after an injection of 0.1 c.c. of 1:10,000 Old Tuberculin of Koch was four plus, with the formation of phlyctens.

Medical examination. The following observations were made: Scrofular facies; cervical glands firm on both sides, more so on the left. Denture incomplete, with many cavities up to grade 4. Granular pharyngitis. Thorax large. Lungs: pectoriloquy extending to the sixth dorsal vertebra. Heart: sounds normal. Arterial tension 12/9 (Vaquez). Abdomen normal. Roentgenogram of chest: both pulmonary fields clear. The horizontal fissure visible (old pleuritis) in its external half. There is no active pulmonary process (Dr. R. Hevia, phthisiologist). Summary: "This patient presents cervical adenitis and signs of tracheobronchial adenopathy (pectoriloquy) which together with radiographic shadow of the lungs, intensely positive tuberculin reaction, facies, and ocular lesion, impose a diognosis of scrofula" (Dr. Emilio Ureta, Pediatric Consultant for the Clinic).

The fact that adults present infantile types of scrofula has already been observed. Adolph Gutman, Professor of Ophthalmology of the University of Ber-

lin, while in Chile, wrote to us after having seen some of our patients: "In the last years of the war of 1914-1918, in which I participated as the medical chief of the German Army, soldiers 20 to 45 years of age presented themselves with scrofula. Many of these persons had in infancy suffered from scrofulous ocular inflammations and had received insufficient food, etc. Some cases were presented in the adult for the first time. This then was the occasion for communications to the Medical Societies of Berlin. After the end of the war we saw the same affection also in adults who had not been in the war. In this ocular scrofulosis of adults phlyctenular conjunctivitis and corneal ulcers were treated."

Thus in these patients there can be no doubt about the etiology of the ocular and general affection. A scrofulosis is involved—that is to say, a glandular tuberculosis—with the consequent toxicosis. This pathogenesis is universally accepted in pediatrics.

In turn the ocular affections of the type studied by us, tuberculin sensitive, are also universally considered in ophthalmology to be tuberculous infections, but the basis of this concept is not a matter to be discussed here.

In fact, for the past 40 years, through the work of von Michel and his school, ophthalmologists have considered tuberculous iritis to be a painless inflammation without any specific bacillary character macroscopically or microscopically (afollicular or anodular iritis). Certain types of keratitis, scleritis, choroiditis, and retinitis are also of the same etiology and modality. For these affections of the adult, ophthalmologists accept the pathogenesis evoked by scrofulous inflammations, that is to say, inflammations secondary to a nodular bacillary toxemia, and, like the pediatricians, go back to tuberculin therapy.

The above-mentioned toxic syndrome, seen with extreme frequency in tuberculous ocular affections, has diagnostic value. As has been said, it follows as the sign of bacillary ocular affection.

When, therefore, I meet ophthalmologists perplexed with a clinical case of an etiology impossible to determine, I try to find the presence of the toxic syndrome. If this is present, without more ado a tuberculous etiology is suspected and I proceed as indicated; in other words, tuberculin treatment is given a trial. This has the value of a positive Wassermann reaction for a syphilitic etiology.

Some examples in which the characteristic toxic syndrome accompanies the tubercular ocular affection are as follows:

Case 2. Margarita M. suffered from tuberculous iritis.[1] Here is how the patient recounted her illness:

"For the past three months I have not even been able to see my hands. For a year I have had severe headaches which did not allow me to sleep; I suffered from insomnia, sleeping only in naps. I had strength for nothing: on serving I had to hold the plate with both hands. I had no energy, working from necessity, from obligation. I have become very thin; formerly my weight was 66 kilos, now it is 44. I looked ill; my employer said I had the face of the tubercular and sent me to a chest physician, who found nothing. I also suffered from periods of severe diarrhea, alternating with constipation. At times I could hear nothing and did not understand when people spoke, as it seemed that they spoke from far away."

This patient submitted to tuberculin therapy. I saw her vision improve, the toxic syndrome disappear, and she gained 5 kilos in weight. Examination for tuberculosis revealed thoracic calcification, nodes in the neck, and a cervical scar from old suppurating adenitis.

Case 3. Victoria C.[1] was suffering from

tuberculous sclero-kerato-iritis, treated unsuccessfully with salversan, bismuth, and the like. She improved with antigenic therapy, which made the headache, insomnia, gastrointestinal disturbances, and chronic rhinitis disappear. She gained 13 kilos in weight.

Case 4. Enrique M.[2] suffered from blindness from bilateral iridochoroidoretinitis. He submitted to energetic antisyphilitic treatment without success and improved under tuberculin treatment. The ocular affection was accompanied by obvious decline in the general condition, which the patient described thus: "About three months before having trouble with my vision, in November, 1938, I had a serious intestinal illness with fever, which obliged me to remain in bed a month. From then on I have not felt well. I have become thin: I now weigh 60 kilos, while previously I weighed 65 kilos. I had no appetite, whereas previously it was very good. I did not sleep well, waking up several times in the night and sleeping only one or two hours at a time. I perspired at night in amounts sufficient to wet the sheets, and suffered from constipation and daily headaches. I had energy for nothing and was always tired." Tuberculin therapy improved vision and banished the toxic syndrome. The patient gained 10 kilos in weight.

It is not necessary to prolong the exposition with clinical records in this connection, but the toxic syndrome is the rule in ocular tuberculosis.

I have already called attention to this in my last communication to the "Annales d'Oculiste,"[3] as it does not appear to be mentioned in the textbooks on this specialty.

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Discussion

Dr. Albert Louis Brown (Cincinnati). The systemic state of a patient with ocular tuberculosis has been the subject of much discussion. Physical findings in this country at least have been quite vague in so far as they suggest general tuberculous infection. Numerous reports from tubercular sanatoria of patients with frank tuberculosis show less than 1 percent having ocular diseases that could be construed as tuberculous. Those with ocular lesions considered to be tuberculous have on the whole been reported to be free from objective and subjective signs of tuberculosis. Thus, it would seem that ocular tuberculosis is an isolated phenomenon caused by some absorption, or more probably a local tissue sensitization that induces local reactions without any systemic signs. I agree with the essayist that if such a patient is watched closely enough some general manifestations are observed. I find the most common to be fatigue or at least greater fatigability. This prominent symptom seems to be directly proportional to the amount of ocular involvement, but not necessarily to the severity of the inflammation. Isolated choroidal tubercles or low-grade retinal inflammation with periphlebitis seem unassociated with any subjective systemic reactions. Uveal inflammation, however, is frequently accompanied by great malaise, headaches, and loss of appetite. These symptoms occur with no demonstrable systemic tu-

berculous infection. In my experience such patients do very poorly if they are allowed to continue to work or are subjected to worry, or nervous strain. Bed rest while undergoing treatment during the early phases of inflammation is as essential as any one therapeutic measure. Testing with tuberculin to determine the optimum dose for treatment must be done with great caution at this stage. Injudicious use of tuberculin may cause any ocular reaction, from a mildly increased inflammation to a profound intraocular exudation. The initial test should be an intradermal injection of not more than 1:100,000 mg. of old tuberculin, or its equivalent and less if the ocular inflammation is severe. The temperature should be charted every three hours, so that a composite picture of the local, focal, and general reaction may be obtained. After the minimum threshold dose is established, cautious increases in dosage may be begun at the optimum intervals. The optimum interval is the time for the local reaction to subside completely plus 24 hours. This is usually a four- or five-day period. As desensitization proceeds, a routine may be established that is most practical for the patient.

VALUE OF GONIOSCOPY IN THE PROGNOSIS AND TREATMENT OF GLAUCOMA*

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Although of recent date, the examination of the angle of the anterior chamber in the living eye has increased considerably in clinical value with the development of new instruments and the systematic and critical study of numerous cases of the varied diseases which affect the structures.

It is not my intention to consider here the value of gonioscopy in all pathological conditions found in this region of the eye, but to confine myself to bringing forward the importance of the aid which this method may render in the clinic for the prognosis and treatment of the several types of glaucoma.

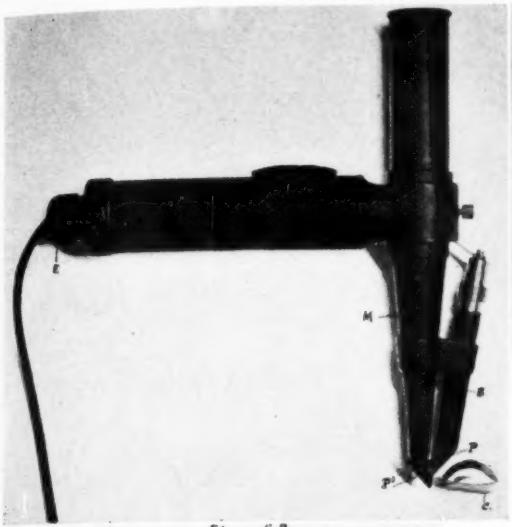
I will describe first, briefly, the methods and instruments used at present for the examination of the angle. To obtain good images and sufficient magnification, two methods are available: (1) the instruments which I have devised and given the name of gonioscopes; (2) the slit-lamp microscope, using it either as a whole or only the microscope head.

1. In several articles[1] I have minutely described the gonioscope and how to use it in the examination of the angle. This instrument (fig. 1) carries a periscope and a concentrated source of illumination. It gives very good images and permits the examination of the sinus or recess of the anterior chamber deeply *on the same side* as the observer stands, when the hemispheric contact glass is used. However, the use of two prisms for lateral observation makes the instrument a little difficult to focus and requires special training. To avoid these drawbacks I have devised a new model which has no prisms and in which the examination of the angle is direct. It gives a clear, side view through the contact glass of the opposite part of the angle, but requires the observer to move around to

* From the Department of Ophthalmology, Columbia University, and the Eye Institute of the Presbyterian Hospital, New York. Read at the first Pan-American Ophthalmological Congress, Cleveland, October 11-12, 1940.

examine the inner limbus on all sides. This instrument (fig. 2) carries a powerful source of illumination, which can be concentrated to a slit and gives magnifications of 10× and 20×.*

I have made it monocular to keep it as handy as the ordinary ophthalmoscope. A binocular field is not so important in viewing the angle of the anterior chamber, where there are only two planes to consider; the last roll of the iris and the corneoscleral wall. Besides, the price of this new gonioscope will be much lower than that of the first model. However, I have no objection to the binocular instrument, my only purpose being to facilitate a widespread use of the gonioscope. In fact a stereoscopic model (fig. 3) has been made by Mr. Kern Larkin and myself, using a lighter model of the ordinary slitlamp microscope and attaching to it the same coaxial illumination that was used in the monocular model.



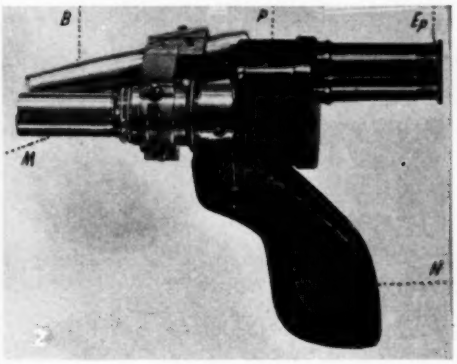


Fig. 1 (Troncoso). First model of gonioscope, showing, C, contact glass; P, prism to deflect the rays of light from the illuminating system, B, into the angle; P', square prism which receives rays coming from the angle and sends them into the microscope, M; E, electric connection from the handle of the instrument into the rheostat.

Fig. 2 (Troncoso). New monocular model of gonioscope for direct examination of the angle of anterior chamber. M, microscope tube and objective; P, redressing prism; Ep, eyepiece; B, illuminating system connected to a rheostat; H, handle.

Fig. 3 (Troncoso). Binocular model of gonioscope consisting of ordinary slitlamp binocular microscope, provided with an illuminating system, B. This is the same as in figure 2, and co-axillary to the microscope; H, handle; Ob, objective; E, electric connection; p-p, prisms; Ep, eye piece.

2. The slitlamp microscope of the large instrument has been used mainly by holding the microscope head in the hands or, as it is very heavy and difficult to keep steady for focusing, fastening it to special stands devised by many observers (O. Barkan, Hartshorne, McLean, and

*This instrument is made by Mr. Kern Larkin, 635 West 165th Street, New York.

others). It is also necessary to use an independent source of illumination, which may be a powerful flashlight or the O'Brien or the Barkan model. The combined price of the microscope, the stand, and the illuminating system makes this instrument quite expensive. It is also cumbersome and difficult to apply to the four sides of the angle.

Dr. Goldman, of Berne, has recently devised a new contact glass with which the angle can be examined with the slit-lamp instrument as a whole, the patient sitting in front of the observer.

Gonioscopy in glaucoma. The glaucomatous process determines, soonor or later, the adhesion of the iris root to the corneal wall, or anterior peripheral synechia. Gonioscopy gives us the opportunity to observe its development in the living eye. Careful and numerous examinations have shown that the changes in the angle in different types of glaucoma are as follows:

In the *acute congestive attack* the examination of the angle is usually impossible on account of the turbidity of the cornea, but in prodromic glaucoma and when the attack has subsided, gonioscopy discloses that the closure of the angle by peripheral synechia does not exist before the acute symptoms, and that even after the attack the angle may be open or only partially closed; that is, peripheral synechia is the result, not the cause of hypertension.

In the chronic congestive type the angle becomes closed in the majority of cases.

In chronic simple or noncongestive glaucoma, on the other hand, the angle is usually open and remains so for a long time, until the advance of the glaucomatous process determines the formation of partial peripheral synechia, which later becomes total all around the limbus. It is especially when slight congestive attacks occur (which sometimes remain unknown, even to the physician) that the angle closes in simple glaucoma.

To give an idea of the condition of the angle in different types and periods of the disease, I will quote from a former paper[2] my own statistics, as follows:

TOTAL OF EYES EXAMINED IN 3 SERIES = 87

Congestive Glaucoma

	Percent
(a) *Acute or chronic with acute or subacute exacerbations*	
angle open all around	26
angle partially closed	46
angle entirely closed	26
(b) *Chronic congestive*	
angle open	25
angle partially closed	25
angle entirely closed	50
(c) *Simple or noncongestive*	
angle open	56
angle partially closed	38
angle entirely closed	5
(d) *Simple with acute exacerbations*	
angle partly closed	50
angle entirely closed	50
(e) *Secondary glaucoma* (different diseases: uveitis, diabetes, occlusion of central vein, traumatic, etc.)	
angle open	61
angle partly closed	15
angle entirely closed	3

The statistics of Werner,[3] Thoburn,[4] and McLean[5] are similar to my own figures.

Prognosis. The prognosis of glaucoma has to be based not on one but on several symptoms. It changes, of course, with the type of the disease. The condition of the angle is only another symptom to be added to the following: (1) the presence, height, and course of hypertension; (2) the reduction of the field of vision and the presence or absence of Seidel's and Bjerrum's scotomata; (3) the existence of an excavation in the disc, its depth, and the color of the optic nerve; (4) the general condition of the patient and the existence of nervous, metabolic, or hypertensive diseases. Along with these four important

data, gonioscopy gives valuable information about the presence of peripheral synechia and also important warnings of the progress of the disease.

Congestive glaucoma. As is well known, in the acute attacks the prognosis depends on the early performance of an operation. In cases of prodromic glaucoma, in addition to the other symptoms, the narrowing of the angle or its partial closure should be considered an indication for proposing an immediate iridectomy. The fellow eye of patients with congestive glaucoma should be carefully examined, as glaucoma is usually a bilateral disease.

In chronic congestive conditions an entirely closed angle is of bad prognosis, and demands not only an iridectomy, but also a fistulizing operation; that is, trephining or Lagrange operations. Cyclodialysis, especially with magnesium implantation, may be tried; it has given good results in many cases.

In simple glaucoma the old idea of continuing miotics for long periods of time should be revised. The curve of the intraocular tension should be drawn from time to time with diurnal and nocturnal observations. Slight symptoms of hypertension should be watched, the patient being instructed to come to the physician when colored halos, blurring of sight, and headaches or violent pains start. In this case the tension should be taken and the eye examined with the slitlamp microscope. This will show injection of the eye, corneal edema, and slight dilatation of the pupil. When there is turbidity of the aqueous with floaters and deposits in Descemet's membrane, uveitis is present, with secondary hypertension. Before and after these attacks gonioscopy may show either an open angle or a partial peripheral synechia. It is my opinion that when the latter appears, and advances, an indication for operation exists.

In a general way we may state that an early operation should be the rule in primary glaucoma.

In secondary glaucoma the prognosis depends on the nature, severity, and course of the original disease. However, gonioscopy may be helpful to show if there is any involvement of the angle.

Treatment of glaucoma. The examination of the angle should be made before an operation is undertaken in chronic congestive and especially in simple glaucoma. If there is a partial peripheral synechia, operations on the limbus will have more probabilities of success and of avoiding complications, if made at the places where the angle is open. This is especially important in cases of trephining and cyclodialysis.

Iridectomy is usually made above to avoid dazzling, which a large pupil almost always produces. When there is already a permanent, complete, peripheral synechia, tearing of the iris root, as advised by v. Graefe, is impossible, as the base of the iris will remain attached to the inner cornea. In these cases a strip of iris is always left above the coloboma.

Other antiglaucomatous operations such as iridencleisis, iridotasis, and the like, will have better results if the presence and place of peripheral synechia are previously detected.

After glaucoma operations gonioscopy is also useful. It will give interesting data regarding the correct performance of the operation and detect complications which the surgeon, examining only the outside of the limbus, can never surmise.

In a previous article Dr. A. B. Reese and I[6] have described, in detail, the complications that may be found after trephining. They can be divided into three groups: (1) adhesions of the pillars or base of the coloboma, or even of the surface of the iris, to the scleral scar; (2) incarceration of one or several ciliary processes into the scar of the operation;

(3) the presence of exudate on the edges or surface of the coloboma and in the angle itself.

The first group is not very important for the future of the eye that has been operated on, although the adhesions of the iris usually close the hole of trephining and destroy the effect of the operation. The incarceration of the ciliary processes may have a bad prognosis, especially if the hole remains patent. The processes may become infected and severe symptoms of inflammation develop immediately or at a later period. Exudate seen with the gonioscope consists in bands of connective tissue or membranes covering the coloboma. These are sometimes so thick and extensive that they obliterate the aperture in the iris or reduce it to a narrow slit. In other cases they may bury the processes in a mass of exudate.

According to my own statistics[6] these complications are more frequent after trephining than is a normal healing.

After cyclodialysis with or without magnesium implantation, gonioscopy usually detects a detachment and tearing of Descemet's membrane and separation of the iris root from the sclera. When the fissure is patent it can be detected by transillumination.

In secondary glaucoma after cataract operation the gonioscope discloses a partial or total obliteration of the angle. The best operation to control tension in this case is cyclodialysis.

Summary

A new model of gonioscope is described, which has no prisms and gives a direct lateral view of the angle with good magnifications (10× and 20×). The instrument carries its own illuminating system, is monocular, and as easy to handle as the ordinary ophthalmoscope. It may be made binocular if so desired.

Gonioscopy shows the development of the characteristic symptom of glaucoma in the angle of the anterior chamber; namely, anterior peripheral synechia. The author studies its frequency in the different types of the disease and its importance on the prognosis and treatment. In congestive glaucoma the prognosis improves when an early iridectomy is performed. The fellow eye should be carefully examined.

In chronic simple glaucoma gonioscopy gives important data which should be added to the other well-known symptoms of hypertension. The old idea of continued treatment with miotics alone should be revised. If slight attacks of hypertension appear, if changes in the field of vision advance, and a peripheral synechia begins to form in one or several sides of the limbus, it is time for an operation. In a general way, an early operation gives a better prognosis in primary glaucoma.

In the treatment of glaucoma, gonioscopy is important before and after operations. Iridectomy, cyclodialysis, trephining, iridencleisis, and other operations have more chance of success when performed at places where the angle is open. The Lagrange operation or trephining opens a permanent channel of outflow, but the second is usually followed by complications. Cyclodialysis may be useful even when there is a peripheral synechia.

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Discussion

Dr. H. Saul Sugar, Chicago: I feel it a great privilege indeed to have the opportunity to discuss a paper on gonioscopy by the pioneer author on this subject in the Americas. Dr. Troncoso has not alone introduced new instruments for gonioscopic study but has correlated the comparative anatomy, the microanatomy, and the clinical appearance of the anterior-chamber angle into a firm foundation of scientific facts.

I have used the new gonioscope and recommend it especially for those who do not have access to a slitlamp microscope.

May I compare with Dr. Troncoso's statistics on the condition of the angle in the various types of glaucoma, a group of 257 eyes with primary glaucoma examined gonioscopically during 1939:

	No. of eyes	*Angle*
Congestive or incompensated glaucoma	17	too shallow
Simple, compensated, or noncongestive glaucoma	240	9 too shallow 215 no synechia 16 synechia (10 total, 6 partial)

Of this group we are particularly interested in those with simple glaucoma, of which there were 240 eyes. Nine were too shallow for observation, 215 had no synechia, and 16 had. Of the latter, 10 angles were totally obliterated by adhesions and 6 partially. The significant point is that those without adhesions were in eyes with early or moderately advanced glaucoma, while those with adhesions had far-advanced or absolute glaucoma. These findings agree with Dr. Troncoso's statements that synechiae are the result of glaucoma, and not its cause. The mechanism in the acute congestive glaucoma in which synechia formation is so rapid is shown by the experimental work of Dr. Jonas Friedenwald of Baltimore. He induced edema of the ciliary body and acute glaucoma in dogs by injecting histamine into the vitreous. Congestion and swelling of the ciliary body are unquestionably chief factors in the formation of synechiae.

In the surgical treatment of glaucoma, gonioscopic control pre- and postoperatively is as important as is examination of the pupillary border and lens under magnification in cataract surgery. In trephining operations the position of the iris root is of fundamental importance. In an eye with peripheral anterior synechia, if the trephining is done at the corneoscleral junction the operation may fail because of prolapse of ciliary processes into the trephine opening. This may be followed by a localized cyclitic reaction which seals off the trephine aperture. Similarly, if it is done in the horizontal meridian of the limbus, even when no angle adhesions are present, there is a tendency to failure because of the narrowness of the limbal lip at the sides of

the cornea. I am also convinced of Dr. Troncoso's correctness in stating that the cyclodialysis operation should not be done in areas of adhesion. This warning has not been heeded by those surgeons who attempt to do the cyclodialysis at the site of adherent pillars in cases of secondary glaucoma following cataract extraction.

Following glaucoma surgery, if the drainage through the newly formed channels is cut off, the hypertension often becomes more difficult to control medically than before surgery because of new adhesions in the angle which formed while the chamber was collapsed immediately following surgery. To determine the site of secondary surgery in cases in which this has occurred, gonioscopy is extremely valuable, particularly after fistulizing operations and cyclodialysis of eyes in which hemorrhages have occurred. I have attempted to avoid many of the latter by decreasing the extent of my spatula sweep during the cyclodialysis operation, using miotics during the postoperative healing period, and avoiding the horizontal meridian where the long posterior ciliary vessels and nerve loops are located.

To determine the prognosis in any eye that has been operated on for glaucoma, especially during the postoperative weeks, when a normal or low tension may be misleading, gonioscopy is helpful. In cyclodialysis the patent cleft is favorable, a closed one unfavorable. In trephined eyes a free opening is favorable and one plugged by ciliary body, iris, or lens is obviously unfavorable. The prognosis depends upon such factors.

AUTOFUNDOSCOPY*

(Autoretinovasoentoscopy; Auto-ophthalmoscopy of Eber; Purkinje figure of Walker)

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In opening a discussion on entoptic phenomena I believe one can do no better than to quote directly from von Helmholtz.[1]

> Under suitable conditions light falling on the eye may render visible certain objects within the eye itself. These perceptions are called *entoptical.* Ordinarily the small opaque particles suspended in the vitreous or aqueous humors or in the crystalline lens do not cast any visible shadows on the retina and are therefore not noticed. This is because the intensity of the light entering the pupil is generally uniformly distributed over every part of it, and therefore, so far as the illumination of the posterior chamber of the eye is concerned, the entire pupil acts like a luminous surface. But when a source of light is a broad surface, no perceptible shadows are produced unless the opaque object is large or else is very near the screen. Now there are certainly some objects in the eye, particularly the blood vessels of the retina, which fulfill the latter condition by being very close to the sensitive membrane behind them, and which are therefore, in position to cast shadows on the retina. But the areas of the retina lying behind the blood vessels are always thus shaded, so that this is their normal condition, and it is only under special circumstances . . . that these shadows become visible.

In 1825 Purkinje had first demonstrated the subjective visualization of the retinal blood vessels. He left a vast maze of experimental findings which, Duke-Elder says, remains largely unsifted. Purkinje described the three basic methods of visualizing the blood vessels: (1) By moving a bright light focused on the sclera, preferably behind the region of the ciliary body. (2) By looking at a dark background, and moving a light in a circular motion close to the eye and slightly off the visual axis. (3) By looking at a bright source of light through a moving stenopeic aperture held close to the eye.

H. Müller was the first to offer a complete explanation of the apparent movement of the blood vessels in relation to the movement of the light. Müller also calculated the distance of the blood vessels from the level of the rods and cones by measuring the distance between the double images of the same blood vessel produced by moving the light a measured distance on the sclera. He found the vessels to be from 0.17 to 0.32 mm. in front of the percipient layers of the retina. Histologically he then found the layer of cones to measure 0.2 to 0.3 mm. from the retinal blood vessels and considered thereby that he had proved that the cones were the sentient layer.

Vierordt in 1856 estimated that the blood corpuscles moved 0.5 mm. per second in the macular vessels.

Again quoting almost directly from von Helmholtz,[2]

> Normally the blood vessels of the retina project hazy shadows on the percipient layer immediately behind them, the length of the umbra being only 4 to 5 times the diameter of the blood vessels. According to E. H. Weber the thickest branch of the central vessel is .038 mm., and Kölliker estimates the thickness of the retina at the macular area as 0.22 mm. Hence it may be assumed that the umbra of any blood vessel does not reach the posterior surface of the retina at all! But when the light enters the eye through a stenopeic aperture in front of the pupil the shadow of the blood vessel is necessarily smaller and more sharply defined—and since the umbra is longer, parts of the retina which were formerly only partially shadowed are now completely shaded while adjacent regions are not shaded at all.

* From the Department of Ophthalmology, Saint Louis University School of Medicine. Read before the Saint Louis Ophthalmic Society, January 24, 1941.

The reason why we do not perceive the shadows of the blood vessels ordinarily is because the regions covered by the shadow being accustomed to darkness are less fatigued than the rest of the retina. But as soon as the position or extent of the shadow varies it falls upon fatigued or less sensitive retina and therefore becomes visible as feeble illumination; and at the same time the more sensitive areas that are normally in the shadow are exposed to the full light and therefore are more strongly illuminated than usual. This is the reason why sometimes, and especially at the outset of the experiment the appearance of the vascular figure may be momentarily that of a bright outline on a dark background, and indeed many persons pay more attention to this bright aspect than to the dark permanent phase. However when the shadow has taken its permanent position the shaded area begins to recover its sensitivity whereas the unusual illumination of the parts that are normally shaded soon fatigues and benumbs them so that the temporary bright effect soon leaves.

There is by no means universal agreement that this is the true explanation why retinal blood vessels are not usually seen, and why they are seen under special conditions.

According to Duke-Elder,[1] "Any object in front of the percipient layer of the retina which has a refractivity different from that of the medium in which it lies can be made visible entopically." He groups entoscopic images according to their origin; first, from any of the media in front of the retina, second, from the retinal blood vessels (placed in one group since so much study has been made of them), and third from the inner layers of the retina itself. Strictly speaking, all of these groups are of course in the ocular "media."

Auto-ophthalmoscopy

In 1853, two years after von Helmholtz invented the ophthalmoscope, Coccius presented the first *auto-ophthalmoscope* with which a skilled observer could see his fundus with his other normal eye. Purkinje, Heyman, Giraud-Teulon, Zelender, Wessely, and Gould, all made apparatus of varying complexity to accomplish the same purpose. Supposedly none of these investigators considered the instruments as of other than academic importance. Since the time of the ophthalmoscope, then, there has been considerable interest in seeing one's own fundus—enough so that elaborate devices have been made to make this possible. Excellent descriptions of some of the instruments are available ("American encyclopedia of ophthalmology," under the title "Auto-ophthalmoscope," v. 1, p. 712.

Autofundoscopy

In 1922, Eber of Pittsburgh published an original one-page description of the startlingly beautiful visualization of the retinal blood vessels by pressing a lighted electric ophthalmoscope bulb against the closed lid and moving it slightly back and forth in any direction. This is merely the modern counterpart of Purkinje's first method of visualizing retinal blood vessels. Eber said, "The optic nerve appears black and the retinal blood vessels also dark and richly branched." He pleaded that others should use and exploit the method. Dr. Edward Jackson added an encouraging footnote but rejected Eber's proposed name, auto-ophthalmoscopy, or auto-ophthalmofundoscopy. The method could more accurately be called auto-retino-vaso-entoscopy, but autofundoscopy is probably the best name for common use.

As far as I can find nothing further appears in the literature until Benjamin Friedman of New York City in 1931 published another one-page article, entitled, "A test for retinal function in cataractous patients." He added nothing to the technique described by Eber. Friedman too described the retinal blood vessels appearing as a black tracery against the red background. He claimed that, "Retinal detachment (in cataractous eyes) may be readily visualized and most

likely this method will prove of value in instances of retinal detachment in which the projection of light is not faulty."

In 1939 and in 1940 Clifford Walker of Los Angeles, under the title "The Purkinje figure as applied to give an absolute and differential test of the presence of separated retina" advised using repeated visualization so that the patient could learn to visualize his retinal blood vessels while the eyes were open. If the patient "fixates upon a screen or perimeter, the character of the branching at various degrees of the field may be approximated." Walker claims that ". . . even in the flattest retinal separation there is no visualization of the retinal blood vessels because the slightest film of edema in the potential space between the two primary retinal layers (crossed only by percipient element tips) will increase the penumbra of vascular shadows beyond recognition." According to von Helmholtz and most of the authorities who have followed, the *formation of the shadows upon the layer of rods and cones* is the thing necessary for visualization. A separation of the retina would not materially affect in any way the formation of these shadows upon the rods and cones unless one of two conditions were present. First, if the retina were far displaced toward the source of light the effect would be as if the source of light itself were broadened; the length of the shadow would be decreased. If the length of the shadow is markedly decreased it might be that the shadow would not reach from the blood vessel to the layer of rods and cones, and therefore the blood vessels would not be visible. However, if this were a considerable factor, autofundoscopy should vary considerably, depending upon whether the light source is placed forward or backward on the sclera, and it does not do so. Secondly, the possibility exists that in detachment the separated retina itself might increase in thickness. As far as I know, no serious attempt has ever been made to show that separated retina swells; that is, separated retina still capable of vision. But if separated retina still capable of vision did swell, the distance between the blood vessels and the rods and cones would be increased. If increased enough, so that the shadows did not reach the rods and cones, the blood vessels would become invisible. Just why Walker considers it logical that the slightest film of edema separating the rods and cones from the pigment epithelium will increase the penumbra of the vascular shadows beyond recognition is not at all definite. Of course, if the pigment epithelium were the percipient layer of the retina no detailed explanation would be necessary. Then edema between the primary retinal layers could quite conceivably lengthen out the shadows formed on the retinal pigment epithelium beyond recognition.

Autofundoscopy, as described by Eber and by Friedman, can be done with any small pocket flashlight. The pencil flashlight made by EverReady Company, to retail at fifty cents or less, does very well.

Technique of Autofundoscopy

Autofundoscopy while giving most beautiful visualization of the blood vessels, particularly of the vessels and capillaries of the macular area, fails to give one localization and proportion. The average patient can see the blood vessels, but he does not know nor can he tell where or how large a lesion is. By showing him many fundus pictures it might be possible that he could localize a retinal lesion. The patient could, of course, fixate the center of a screen at arm's length and in semidarkness and, projecting any lesion visualized upon the screen, draw its outline. But in actual tests upon myself such a procedure was exceedingly difficult

so that grave doubt is aroused that such a procedure can be of any clinical value. Again, a cataractous patient, for instance, probably could not do this.

Therefore, two ordinary showcase tubular lamps with linear filaments were mounted in a small box at right angles to each other (fig. 1). The patient now

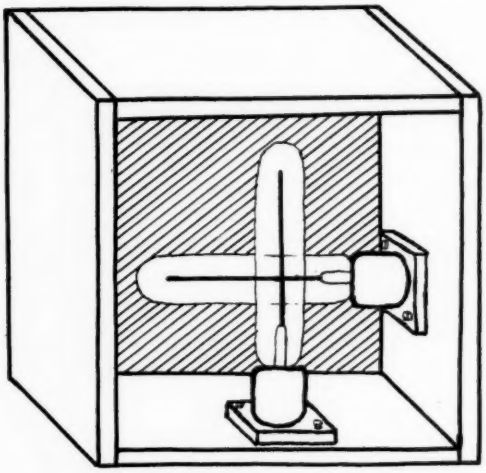

Fig. 1 (Drews). Apparatus for inducing crossed-filament afterimage.

was asked to fixate the center of the cross formed by the lighted filaments, for 15 to 30 seconds. With autofundoscopy, the area of the retina* bearing the afterimage first shows a finely granular appearance, distinguishing it from the rest of the macula. After a second or two a striking negative afterimage of the filaments appears, usually of a dark greenish-black color. It is noticeable that the width of the afterimage is considerably greater than the width the filaments appeared to be. The afterimage goes through variations as do other afterimages, becomes bright and fades, disappears and returns, changes color and halo-color. As much as thirty minutes after the afterimage has faded from view autofundoscopy may again bring it back. It is well known that in some individuals the primary afterimage can be reinduced by a secondary stimulus as much as two hours after exposure.

The cross of filaments could be greatly refined with perhaps some improvement in technique. A single lamp shaped in the form of a cross and with two crossed filaments could be made up. Such a lamp, however, could not be used in determining the presence of anomalous retinal correspondence (Bielschowsky), whereas this homely apparatus serves both purposes very well. Studies with a cross of light formed by illuminating two narrow crossed slits with an instantaneous flash of light (as with the photo-flash lamp) are to be done later to see if the apparent widening of the filaments in the afterimage is due to slight movements of the eyes while fixating, or if this is due to retinal induction.

The filaments of the lamps used measure 76 mm. in length. At 1 meter a 1-degree target measures $\frac{2\times1000\times\text{Pi}}{360}=$ 17.5 mm. The 76-mm. filaments used at 1 meter therefore present a $\left(\frac{76}{17.5}=4.34\right)$ 4⅓-degree object. Disregarding any possible increase in the length of the afterimage due to retinal induction or due to small movements of the eye, the crossed filaments at one meter give rise to a 4⅓-degree afterimage. If the patient fixates the center of the crossed filaments, the center of the crossed afterimage marks the center of the fovea. Thus with this very simple expedient both localization and proportion are given to autofundoscopy!

The cross of filaments is purposely

* For discussion as to whether the imprint of the afterimage is made upon the retina or the visual centers, see Duke-Elder, volume 1, page 963. We will assume that the retinal theory is accepted.

made "off center" so that two arms of the cross are much longer than the other two. If the patient draws this cross in proper proportion it is reasonable that he may also draw any central retinal lesion in proportion. The excentric cross thus gives the oculist a ready measure of the patient's sense of proportion.

Afterimages

The fact that visual activity continues after stimulation of the retina has ceased has been known since very early times (Duke-Elder[2]). The afterimages produced in this way were known to Aristotle. At the time of Peirsec (1634), they were regarded as a magician's trick. Kircher (1646) considered them to be caused by light sent out again by the eye after it had been absorbed. Newton 1689) regarded them as psychical in origin. They engaged the attention of Goethe (1819); but the first comprehensive study was done by Purkinje (1819), Plateau (1833), and Fechner (1838).

Induced afterimages

If an afterimage is produced and then the eye exposed to a second larger stimulus (Duke-Elder[3]), the first negative afterimage lies within the positive image of the second stimulus so that the first afterimage is surrounded by a halo of its complementary color. This condition provides material for the most complex reactions, for we are dealing with responses that vary according to the nature and intensity of the light, and involving two distinct retinal areas with different latent periods and different types of activity.

An afterimage may wax and wane in the most irregular manner, the responses tending, however, to be rhythmical. Successive afterimages may show a very variable color series of extraordinary beauty, richness, and saturation.

Binocular afterimages

Bielschowsky has recommended that the patient fixate the center of a horizontal filament of light with one eye, then fixate the center of a vertical filament with the other eye. Now the afterimages will form a cross provided there is normal retinal correspondence.

Parinaud (1882) investigated the effect of a secondary stimulus in the one eye when the primary stimulus had acted only in the other eye. He found that secondary stimulation of the other eye markedly influenced the afterimage of the first eye.

Methods of determining retinal sensitivity in cataract

According to Kronfeld, "Experience has shown that an eye in which there are dioptric obstacles such as corneal scars, membranes, cataract, etc., can still recognize the light of a candle at 6 meters, and can also without any searching movements recognize the position of the light everywhere in the visual field at one meter. In other words, the minimum visual acuity of any eye with intact retina and conduction pathways is candle-light perception at six meters, and candle-light projection at one meter in a dark room." Fuchs says, "Quantitative perception of light centrally is not disturbed by even the densest opacity," and he gives the same minimum vision as does Kronfeld. But Daniel Kirby and Gifford, among others, state that there is no absolutely accurate method of determining *macular* function so that the presence of a blind macular area may be revealed only after cataract extraction has been successful and will account for failure to obtain good vision in a certain number of cases. Duke-Elder[4] mentions that the strength of an electrical current necessary to stimulate the retina is the same whether it be

passed anteroposteriorly or posteroanteriorly, but that in retinal disease the necessary currents do vary, *and that this fact may be of clinical value.* This would be a complicated way of determining retinal function, indeed. Duke-Elder[5] devotes a half page to Eber's autofundoscopy, as does also Spaeth, and both recommend its use.

Case reports

Case 1. Mrs. J. B. C., a young woman, has beautiful, densely medullated nerve fibers contiguous with and above the disc. With autofundoscopy she localized a zone of whitish color with frayed edges, three diameters of the afterimages of the crossed filaments (13 degrees) to the right side and situated one diameter below the horizontal, corresponding with the position of the medullated nerve fibers.

Obviously, if von Helmholtz's explanation of the occurrence of afterimages of the retinal blood vessels is correct, the retina beneath the medullated nerve fibers must be sentient. The patient saw the *white area,* not a white edge (which perhaps would not be so easy to explain). Autofundoscopy could be of clinical value in a case in which a small patch of thinly medullated nerve fibers situated away from the disc may be difficult to diagnose with certainty, especially if the paracentral area is involved.

Case 2. Mrs. M. W., a white woman, aged 75 years, came to Firmin Desloge Clinic (1937) with mature senile cataract in the right eye, and incipient cortical cataract in the left eye. She had marked hypertension and generalized arteriosclerosis. Projection of light in the right eye was excellent, and corrected vision of the left eye was 20/60, which about corresponded with the degree of lenticular interference. The fovea of the left eye appeared normal. There was marked retinal arteriosclerosis. With autofundoscopy she said there was a central blank area in the right eye. Therefore considering the bad general condition I advised against operation. The eye resident promptly had the patient seen by another member of the staff who, knowing nothing about the autofundoscopy findings, advised cataract extraction. After a very successful operation the patient was found to have a central areolar choroidal degeneration in the right eye, and her best vision with correction was 10/200. She had accepted cataract operation with the expectation that reading vision would be restored! Her disappointment was of relatively short duration for about nine months later she died of cerebral accident.

Case 3. E. K., a white male, aged 71 years, was first seen in 1936. He had primary simple glaucoma in the right eye, and retinal arteriosclerosis of rather marked degree in both eyes. There was no evidence of glaucoma in the left eye, but retinal hemorrhages, and an arcuate paracentral angioscotoma below. Most of the retinal hemorrhages of the left eye were in the zone of retina above the fovea corresponding to the scotoma. Several times a widespread edema of this area was also observed. Over a period of three years there was never any evidence of glaucoma in the left eye; no Rönne step was present; the pressure was about 18 mm. Hg (new Schiötz); on several occasions dilating the pupil with euphthalmine and cocaine, 1 percent of each, failed to increase the tension, and pilocarpine miosis also failed to reduce the tension. However, he was instructed to use a small drop of pilocarpine 1/240, oil solution, in the left eye at bedtime.

In April, 1939, he came in with the vision of the left eye reduced to the ability to see the direction of hand movements at 1 ft., due to vitreous hemorrhages (and retinal hemorrhages?). The

pilocarpine was stopped. Vitreous hemorrhage has persisted since then, preventing any ophthalmoscopic view, and best visual acuity since then has been 3/200. In June, 1939, the patient developed acute noncongestive glaucoma, left eye (the eye heretofore free of glaucoma), the pressure rising to 60 mm. Hg (Schiötz) and requiring 6 weeks to be controlled. In October, 1939, a second attack of noncongestive glaucoma developed, the pressure rising to 75 mm. Hg (Schiötz), and the iris becoming a light green color. The intraocular pressure gradually receded over an 8-week period, and has remained normal since December, 1939.

In February, 1940, autofundoscopy, of the left eye, showed a few central hemorrhages still limited to the upper half of the macular and perimacular area. Although the vitreous still contains old blood preventing any view of the fundus, or charts of visual fields, autofundoscopy shows that the central retinal area remains sentient and, indeed, probably unchanged.

Case 4. Mr. L. L., white, aged 59 years, presented a small oval slate-blue choroidal lesion one-third disc diameters high and two-thirds disc diameters wide, situated just between disc and fovea. Aside from moderate vitreous exudate the eyes were otherwise normal. Autofundoscopy failed to show any central lesion, and elaborate central-field studies also failed to show any abnormal scotoma, indicating that the lesion is really choroidal. A lesion wholly confined to the deeper layers of the retina will ophthalmoscopically show no interference with the retinal blood vessels. Such a lesion could be mistaken for a choroidal lesion. Failure to visualize such a central lesion almost certainly places the lesion behind the layer of rods and cones; practically, in the choroid.

Case 5. Mrs. R. R., white, aged 36 years, stated that eight years ago she lost her vision gradually, over a period of three to four days, three weeks postpartum (first baby). She was treated by an oculist and made a gradual recovery over four to six weeks. Visual acuity with or without correction was 24/19. Ophthalmoscopically, widespread drusen-body formations extremely dense but discrete and small throughout the fundi were seen. With autofundoscopy the patient saw only the uniform red background and the blood vessels, without any visualization of the tiny thickenings in the lamina vitrea. Such a test is of real value, for in the presence of widespread drusen formations or very large drusen the retinal nutrition from the choroid may be interfered with, producing secondarily small scotomata; also, any small retinal lesions could easily be overlooked. No comment is made regarding any possible significance of history.

Case 6. Mr. O. S., white, aged 67 years, noted some slight uncertainty in reading vision two to three months previously. Two days before consultation a positive central scotoma in the rough outline of an aeroplane appeared before the central vision of the right eye. With best correction right eye vision was 24/75 excentric; left eye vision, 24/24 plus. Ophthalmoscopic examination showed moderate arteriosclerosis right and left, with a white-gray retinal plaque in the macular area, the exact shape as drawn later by the patient with autofundoscopy (except, of course, turned around and upside down). Stereocampimetric fields were not satisfactory. With the perimeter the peripheral fields were normal in outline (5/350 white) and a central scotoma, extending 5 degrees downward, and 1 to 2 degrees elsewhere from the fixation point, was demonstrable (2/350). Localization of the macula with the afterimage of an ordinary 100-watt naked filament

lamp in both eyes was as indicated in the drawing (fig. 2). The patient was given nitroglycerine, 200 mg. thrice daily, by Dr. C. Kleinschmidt and after two weeks the vision was 24/48. After four more days with sodium nitrite, 100 mg. thrice daily by mouth, the vision of the right eye was 24/30 (and that of the left eye 24/15!!), but the central scotoma, right eye, still seemed to be the same size except that a narrow tongue around the macula had been uncovered. Ophthalmoscopically there was no change. The patient then left the city but did not report to the ophthalmologist to whom he had been referred.

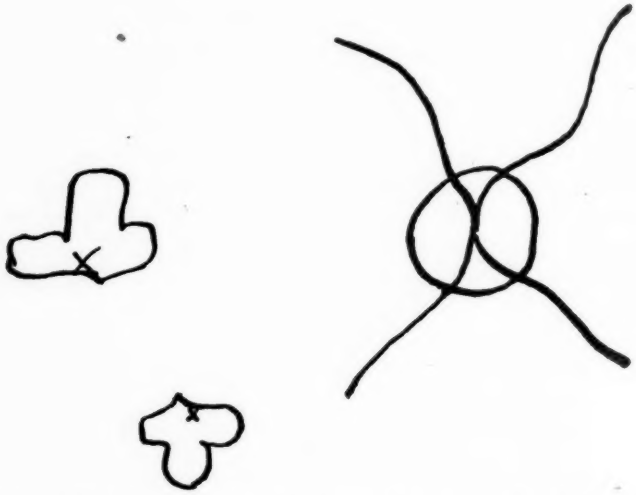

Fig. 2 (Drews). Case 6. Diagram of central lesion, right eye. The center of the X marks the fovea as localized by the patient, using the afterimage of an ordinary 100-watt naked-filament lamp. The lower part of the figure is an exact copy of the patient's drawing of his own central retinal lesion as seen by autofundoscopy.

Autofundoscopy in this case was far more accurate than central-field studies in measuring the field defect, and of course tremendously easier, and more interesting both to me and to the patient.

Case 7. Mr. S. R., aged 63 years, presented in the right eye numerous small retinal hemorrhages over the posterior pole, with a macular hemorrhage one-third disc diameters in size squarely placed over the fovea, marked retinal arteriosclerosis, and central vision reduced to 24/75 excentric. With autofundoscopy the patient drew the central retinal hemorrhages, corresponding fairly well with the ophthalmoscopic picture. This patient was literally obsessed with the idea that the proper lens placed before the eye would restore the reading vision, and he was very insistent because the other eye had an amblyopia ex anopsia, reducing its visual acuity to 24/60. Autofundoscopy and comparison with the sketch I made of his fundus convinced him in a few seconds that the poor vision of the right eye could not be corrected with glasses. Incidentally autofundoscopy can be of great value in convincing the patient that the oculist does speak authoritatively. Second, in this case autofundoscopy showed that the macular area not involved with hemorrhage was still sentient. This information could be obtained otherwise only by very careful field studies; indeed in this case probably could not have been obtained even then because central fixation would have been so uncertain.

Case 8. Mrs. M. C., white, aged 55 years, noted a central red-brown spot before the left field of vision one week before consultation. Visual acuity of the right eye was normal, of the left eye 10/240 excentric. Ophthalmoscopic ex-

amination of the right eye showed marked arteriosclerosis with strapping of the veins, moderately increased light reflex, and moderate variations in caliber. The lower temporal artery appeared almost like a silver-wire artery. The left eye showed similar blood-vessel changes and in addition a dark blue-red dense hemorrhage, two-thirds disc diameters high (supero-inferiorly), and one-half disc diameter wide, with a thin hemorrhagic area beneath it. The center of the ophthalmoscopic field was definitely toward the temporal (left) edge of the lesion when the patient fixated the ophthalmoscope light. With autofundoscopy the patient drew the central lesion.

With the crossed filaments the patient saw only a reddish blurr of light through the hemorrhage. Therefore the patient was asked to fixate the center of the cross with the normal right eye, and the afterimages of the normal eye were then fortified with autofundoscopy in the dark room, after which she could see the cross as an after image "astoundingly well." Now *with autofundoscopy of the left eye the patient visualized the central lesion of the left eye and the "afterimage of the right retina," simultaneously.* This she was able to do very well. Her drawing of the cross and lesion is shown with a sketch of the fundus lesion (fig. 3). If we assume that there is normal retinal correspondence then the center of the afterimage of the right eye must localize the fovea of the left eye, or, in other words, the left fovea must be at the point indicated by the center of the cross! Also, the height of the lesion about corresponds to the height of the cross, which would correspond to a 4-degree plus scotoma (in height). With the perimeter, having the patient fixate her own finger tip, a central scotoma about as indicated could be outlined only with considerable difficulty because of the uncertainty of fixation

Several years ago tests made on similar cases with the stereocampimeter, when the normal eye was used for fixation, were definitely less accurate than monocular or even finger fixation with the poor eye. In this case with the afterimage in the normal eye to mark the fovea of the abnormal eye and autofundoscopy of the abnormal eye there is no known possi-

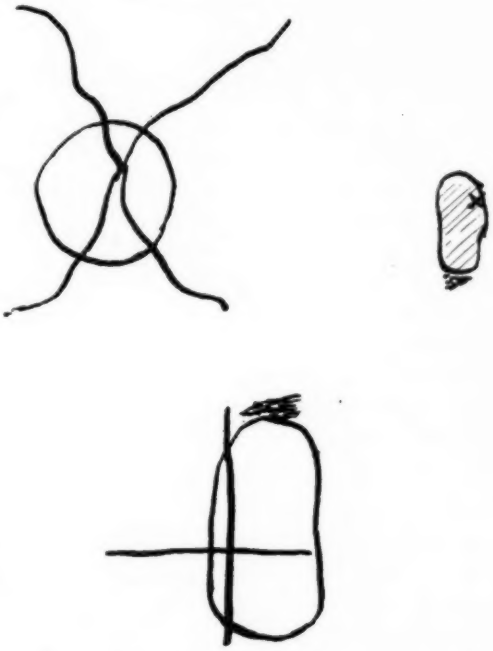

Fig. 3 (Drews). Case 8. Diagram of central retinal lesion, left eye. The center of the X marks the fovea as localized by autofundoscopy combined with visualization of afterimages of the crossed filaments seen by the other eye. The lower part of the figure is an exact copy (slightly reduced in size) of the patient's drawing of the central lesion as seen by autofundoscopy and the afterimage of the crossed filaments seen by the other eye.

bility but that the foveal localization of the abnormal eye is absolutely accurate so long as there is normal retinal correspondence. Not only are we able to measure the scotoma with autofundoscopy in this case, and localize the macular area, but also we are able to do it much more quickly, easily, and, I believe, more accu-

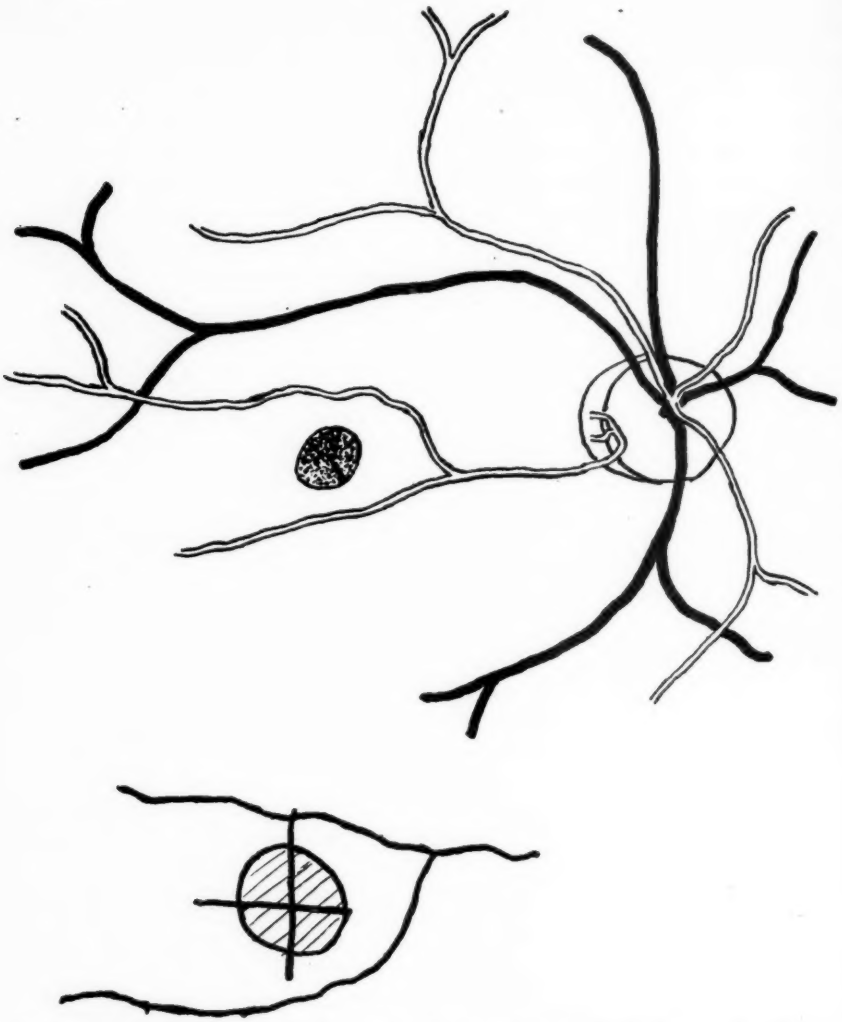

Fig. 4 (Drews). Case 9. A very careful sketch of the blood vessels of this eye was made because it was thought that the cilioretinal artery might have some connection with the position of the fovea. The arteries are represented by double lines; the veins are solid. A narrow zone of choroidal defect forms a temporal crescent. The cilioretinal artery arises from two arteries that pierce the sclera in this crescent. The central lesion is sharply outlined, slightly granular, and the same color as the veins (even in the "red-free" light of the Giantscope). The lower figure is an exact copy of the patient's drawing of the afterimage of the crossed filaments, the central lesion, and the central portion of the cilioretinal artery.

rately than we can by field studies. Of course the patient must have a sense of proportion; the lady in this case used to paint china.

Even if the retina beneath the hemorrhage were destroyed we could still measure as aforedescribed because the patient will note the area where red background is deficient. With such binocular technique the conflict of the visual fields is rather evident, the afterimage and the view of one's own fundus waxing and waning alternately. With a little practice this is usually overcome. The afterimage itself

also waxes and wanes somewhat, but in visualizing an afterimage of one retina simultaneously with autofundoscopy of the other there is obviously the same phenomenon as one sees in conflict of the fields.

Case 9. Mr. W. K., white, aged 60 years, was first seen in May, 1939, when he presented a central retinal lesion as shown in the drawing (fig. 4). A large cilioretinal artery also was present as shown. Considerable freely floating vitreous exudate blurred the fine details slightly but a really good view was obtainable with the pupil dilated. Visual acuity was 20/19, the form field normal, and a relative central scotoma with 1 mm. green target on the stereocampimeter was barely demonstrable, measuring about 1½ degrees on all sides of the fixation point. With white, red, yellow, and blue objects no scotoma could be demonstrated. The patient has been watched since then, and has shown no change in vision, field, or fundus. The left eye has been blind for eight years from chronic uveitis.

In the summer of 1940, an attempt was made to see the retinal lesion with Dr. H. R. Hildreth's red-free binocular ophthalmoscope, but the vitreous exudate blurred details so badly that the lesion could not be seen except as a dark haze. Attempts to measure it with the Keeler ophthalmoscope graticule also failed. Considerable doubt has been expressed by various ophthalmologists to whom I have shown this patient that the lesion is central at all. However, no one has been able to identify the fovea. When the patient is asked to fixate the ophthalmoscope light the lesion appears to me to be in the center of the ophthalmoscope field. Also, according to the drawing, the lesion appears to be on the same level as the lower edge of the disc. With central-field studies the upper border of Mariott's scotoma actually is on the same level as the fixation point. From all these findings the lesion must be in the horizontal plane and therefore very near the fovea.

The patient was asked to fixate the center of the crossed filaments, which were placed one meter from the eye. Previous tests with autofundoscopy repeatedly had shown excellent visualization of the central lesion as a dark-red oval. The afterimages of the filaments were then drawn by the patient in relation to the lesion as visualized by autofundoscopy. The lower drawing is the patient's sketch of the lesion and afterimage. It will be noted that he drew the lesion squarely over the center of the cross. The lesion as outlined by the patient measured approximately two thirds of the diameter of the filaments. At 1 meter the filaments used formed a 4⅓-degree object. Since the lesion seen by autofundoscopy covered two thirds of the afterimage, the lesion might give rise to ($2/3 \times 4\frac{1}{3} = 26/9 =$) a 3-degree scotoma. This exactly corresponds to the measured relative scotoma!

Autofundoscopy in this case proved the lesion to be squarely placed over the fovea and measured the lesion certainly as well and immeasurably more easily than it could be done with field studies. It did not show how relative the scotoma is. That the scotoma is relative may be inferred from the patient's description.

Discussion

A great number of interesting problems suggest themselves. Autofundoscopy and dynamometry combined provide a very ready means of determining what external pressure will produce collapse of the macular capillaries. As far as I have been able to tell on myself the whole fundus fades out almost simultaneously, which may merely mean that the pressure

in all the capillaries is the same. In the near future I hope to be able to investigate this.

Again, how would a central flat detachment of the retina appear? Could the patient himself follow the clinical course of the detachment? Is autofundoscopy reliable in detecting detachment; that is, could the best observer always pick up a central detachment? Contrary to Walker, and Friedman, I believe that *as long as the sentient layer of the detached retina functions sufficiently the patient can visualize his own retinal blood vessels.* I have seen two cases of monocular detachment involving the central fields in which the patient thought both fundi looked alike with autofundoscopy. However, both patients had 30- to 50-degree fields with a one-degree test object. Obviously, if the detached retina loses function to a degree that the blood-vessel shadows are too small to be perceived, the blood vessels will not be visualized. The two cases already mentioned demonstrate that such loss of visual acuity may not occur while the one-degree test object is still visible. If retinal function is lost gradually the smaller retinal blood vessels will fail to be visualized first; later not even large blood vessels may be visualized. Theoretically, the retina could be blind to its own retinal blood vessels by autofundoscopy and still perceive a one-degree test object on the perimeter, since the shadows of the blood vessels are very much smaller than one degree *in width.* In attempting to correlate what sized test object would be just invisible if the retinal vessels were just invisible we must remember that one can see telegraph wires a great distance away. The width of the image of the wire must be very small indeed. One might suspect that the same phenomenon might be encountered in the visualization of the retinal blood vessels which produce long linear images. If the smallest diameter of the image was the deciding factor as to whether or not the image is visible, then with round test objects, the image of which would be the same diameter as that of the retinal blood vessels, we should find an absolute scotoma when autofundoscopy fails to visualize the blood vessels. But really, since long linear images are so highly visible we actually expect to find cases in which such test objects would be quite invisible while autofundoscopy still produced good visualization of the vessels. In other words, we should expect better visualization of the long linear images of the blood vessels than of the image of the round test object the same width as the image of the blood vessel, or even indeed greatly larger than the width of the image of the blood vessel.

Why is it necessary to produce mild pressure of the lamp against the lid? Pressure phosgene apparently has no part in visualizing the blood vessels with autofundoscopy. Does the pressure reduce the blood in the lid, allowing more light to pass?

What rays pass through the lid, conjunctiva, sclera, and ciliary body or choroid? To what is the red background upon which the blood vessels are seen due? According to Duke-Elder,[6] "If the red background is due to capillary blood in the retina then the fovea should not be red." And again (Duke-Elder[7]), "Rivers attributed erythropsia to the blood in the anterior layer of the retina, a theory which is untenable because it would entail the absence of erythropsia in the macula." Autofundoscopy with nearly pure spectral light, using Dr. W. E. Shahan's discs of colored glass with a specially powerful Shahan lamp, failed to show any view with the blue glass. This was very disappointing since I had hoped to be able to see the retinal circulation. With the green glass the blood vessels

appear very dark and the fundus very pale. With the amber glass an interesting foveal reflex is seen, but nothing else. With the red glass practically the same appearance as without it is seen. No attempt was made to use these nearly pure lights in clinical cases. I believe the overlay of red color in autofundoscopy is not due to retinal capillary blood, but that most of the rays except the red rays are filtered out by the passage of light through the eyelids, sclera, and choroid (just as if white light were passed into a dark box through red glass), and that therefore the fundus appears red. In my experience the blood vessels also appear red, and all patients upon whom the method was tried described the vessels as a dark or red color. This does not agree with either Eber's or Friedman's views.

The phenomenon of flicker has little to do with autofundoscopy, for the simple reason that the movement of the light is far too slow and that the speed of the movement need not be changed with varying degrees of illumination.

A considerable number of patients with central retinal or retinochoroidal lesions have been studied with autofundoscopy over the last three years. In no case did the patient think the pathological macular area looked in any wise nearly the same as the normal macular area of the other eye. In one case of binocular macular lesion with absolute scotoma the patient readily outlined the two lesions.

Since all macular or central lesions have been readily discoverable, I believe I have proved that autofundoscopy is of clinical value in determining the normalcy of central fields in the presence of mature cataract. About central retinal detachment no statement can be made, but I suspect that autofundoscopy will fail in a goodly number of cases. Case 9, however, shows that even the visualization of a central lesion does not certainly indicate that the central vision is necessarily defective. I believe this lesion to be an exceptionally rare condition, however. Hope may be entertained that eventually this eye will be examined histologically.

Both Eber and Friedman said that in cases of amblyopia ex anopsia there was a dark spot in the foveal area. Certainly this is not true for low degrees of amblyopia ex anopsia (vision of 20/60 for instance), but in high degrees all eyes tested showed the phenomenon. The central dark spot corresponds with the relative central scotoma that is present.

In creating a 4-degree afterimage it is very noticeable, in my own eyes at least, that the dull-gray haze of Marriott's scotoma is only about one third to one half of this diameter; that is, only one to two degrees of the field instead of 6 degrees! Duke-Elder states[8] that it is of considerable importance that when large areas of stimulation are used the contour of the afterimage is continuous across the blind spot. While the red background has not been considered as a possible afterimage this may be the case. At least, this observation with autofundoscopy is of fundamental importance.

Concerning the color of a central retinal lesion as seen by autofundoscopy, little can be said now. Unfortunately, no special attention was paid to this point. It is, however, my impression that the great majority of patients with absolute scotoma describe the area as a dark green-black color. The failure of subjective visualization of Marriott's scotoma raises a question of whether or not a congenital lesion will also be a disproportionately small more-or-less blank area. If so, this fact could be of value in distinguishing acquired from congenital scotomas. No suggestion is made, however, that this actually is the case.

It is hard to realize that the afterimage of the crossed filaments is only 4⅓ de-

grees in size; it is hard to realize that the central 10 to 15 degrees of the fundus appear so predominant. In this connection it must be understood that the visual acuity of the retina decreases progressively as one leaves the fovea. A very rough but very usable approximation is that the visual acuity of any small retinal area equals the reciprocal of the number of degrees of eccentricity, so that a retinal area 14 degrees eccentric has approximately a visual acuity of only one fourteenth, and so on. More than 20 degrees eccentric the visual acuity of the retina falls off even more rapidly. Hence one should not expect to see peripheral retinal blood vessels with perfect clarity for the simple reason that the sentient layer beneath these peripheral blood vessels has very poor visual acuity. Perhaps for that reason also the central capillaries appear exceptionally richly branched. If peripheral blood vessels do have these capillaries they are not discerned by autofundoscopy. For all these reasons peripheral retinal lesions are easily overlooked by autofundoscopy, and the more peripheral they are the more easily they can be missed. But, after all, the more peripheral the retinal lesion is located the less important is it to determine its presence, unless the lesion is a progressive one.

The proper way to evaluate autofundoscopy as a means of determining retinal function in the cataractous eye is to study results of autofundoscopy in eyes in which the retinal condition is known. Only in this way can we find what shortcomings the method may have. Until a great deal more of this basic "physiological" work is done autofundoscopy should not be depended upon as a sole criterion for cataract extraction. Certainly before one uses autofundoscopy in the cataractous eye he should have tried the method extensively in cases in which the retinal condition is known. The cases of cataract in which autofundoscopy can be of real value are necessarily few; that is, the cases in which autofundoscopy findings will determine whether or not cataract extraction is to be done are necessarily few. In cases in which the one eye is clear enough for ordinary use and the other has excellent projection of light, and in which the patient wants the operation only to have good reading vision restored (as in case 2) autofundoscopy may avoid a good prognosis. Also in cases in which projection of light is faulty but autofundoscopy shows normal central vessels, an operation may be undertaken with the hope that good central vision may be restored. Of course, here it is to be remember that retinal detachment may be present. In the great majority of cataractous eyes the presence of good light projection will be sufficient indication for cataract operation, as always heretofore.

Autofundoscopy is of great psychological value. When one shows a patient who has monocular cataract—who has lost his vision and hopes that somehow it may be recovered—that *the "blind" eye can see* its own blood vessels just as well as the normal eye sees its blood vessels, he will gain his patient's gratitude. The patient is convinced that after all his vision is still there, that it only has to be uncovered. While this is a commonplace to us it is by no means so to the patient, as witness the number of patients who after successful cataract extraction are really astonished that vision is restored. As already mentioned, autofundoscopy of a retinal lesion has also great psychological value. One patient assured me he had never believed the ophthalmologist really could see into the eye with the ophthalmoscope until I drew the lesion he had seen by autofundoscopy, confirming his personal observation.

Conclusion

1. Autofundoscopy properly used and interpreted is of great clinical value.

2. In central retinal lesions autofundoscopy combined with visualization of afterimages of crossed filaments of light is of practical value in measuring the central lesion, and is as accurate as the patient's sense of proportion. Apparently priority may be claimed for this procedure.

3. The afterimages of one eye may be used to measure a central retinal lesion and to locate the foveal area of the other eye. Apparently, priority may be claimed for this procedure also.

4. Retinal detachment is one condition in which autofundoscopy may be entirely unreliable.

5. Pure choroidal lesions and lesions localized strictly to the inner layers of the retina are easily differentiated, if centrally located.

6. The central and paracentral areas are very easy to investigate. No claim is made that lesions outside the central 10 degrees of the retina may be reliably investigated in the average patient by this method. No claim is made that autofundoscopy replaces quantitative central-field studies. Case 9 proves that it does not.

7. Accentuation of the afterimage with autofundoscopy may be considered as an induced afterimage effect. Such accentuation has not to the writer's knowledge been described before.

8. A unique foveal lesion is described (Case 9).

9. The scotoma of the optic-nerve head appears very much smaller with autofundoscopy than it actually is. This is of fundamental physiological importance.

10. An immense amount of work remains to be done on this subject.

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ORTHOPTIC TRAINING AND THE SURGICAL CORRECTION OF STRABISMUS*†

A COMPARATIVE STUDY OF BINOCULAR VISION IN 324 SURGICAL CASES WITH AND WITHOUT ORTHOPTIC TRAINING

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The value of orthoptic training in conjunction with the surgical correction of strabismus has been well demonstrated by numerous observers.[1–14] Lyle[15] is of the opinion that orthoptics must be used as an adjunct to the optical and surgical treatment of strabismus rather than as an independent form of treatment. It has been stated by Guibor[16] that fusion training has a definitely favorable influence on most types of strabismus, even though the angle of squint does not entirely disappear. According to Davis[17] it is a valuable and essential aid to the cure of squint. In a study of 218 patients Bressler[18] noted that the end results are considerably influenced by preoperative and postoperative orthoptic training. Hicks and Hosford[19] are of the opinion that orthoptic training is of great value in developing fusion but that it has a limited value in straightening crossed eyes. In a comprehensive review of the recent literature, McAlpine[20] stated, that although "there is considerable unanimity of opinion in published reports as to the general worth of orthoptics, . . . there is a need for careful reports of the results obtained." We agree with this statement and, therefore, a study of binocular vision has been made in 324 patients operated upon at the New York Eye and Ear Infirmary from 1938 through 1940.

In this analysis no attempt is made to report the incidence of esotropia, exotropia, hypertropia, refractive errors, age of the patients, duration of the squint, familial history, and certain other data. This study is limited to a determination of the effect of preoperative and postoperative orthoptic training on binocular vision as compared with surgery alone. However, the routine examination of the patients included a complete history, cycloplegic refraction, ophthalmoscopic examination and visual acuity with and without correction of errors of refraction. The muscle balance at 6 M. and 25 cm. in the primary position and in the six cardinal positions of gaze was estimated with and without correction of errors of refraction, using the screen test with prisms and screen-Maddox rod test. The near point of convergence and the punctum proximum were noted in all cases. When indicated, central fields on the tangent screen, diplopia chartings, and fields of monocular fixation were studied. The synoptophore was used to estimate retinal correspondence, the grade of binocular vision and amplitude of fusion. Other tests employed were the Worth four-dot test, the Tschermak test, and tests of fatigue of accommodation and convergence, using the ophthalmic ergograph.

To the patients receiving orthoptic training, daily carefully administered preoperative and postoperative treatments were given. Surgery was advised to correct the actual deviation in the majority of cases only after (1) accurate correction of ametropia; (2) amblyopia had

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been overcome so far as possible; (3) normal retinal correspondence was present; (4) suppression had been overcome and (5) some degree of fusion was present. However, operation was advised in 25 percent of the patients after orthoptic treatment had not altered the anomalous retinal correspondence and in 30 percent of the patients in whom fusion could not be developed.

The patients were divided into three groups. In group I there were 144 patients, who were operated upon, but received no orthoptic training. This group was composed of several types of concomitant squint. Group II consisted of 83 patients who received orthoptic training after surgery. In group III there were 97 patients with several types of concomitant squint to whom preoperative and postoperative training was administered. In groups II and III, the main purpose of orthoptic training was to correct amblyopia, anomalous retinal correspondence, suppression, and finally, to increase the amplitude of fusion.

Deviation before and after operation

In 63 percent of 227 patients (groups I and II) who received no orthoptic training before surgery, the deviation measured between 41 prism diopters and 60 prism diopters (table 1). Of the patients in group III who received orthoptic treatment, 53 percent continued to have between 41 and 60 prism diopters of squint after the preoperative orthoptic treatment.

Although in the smaller deviations the actual squint may be somewhat reduced preoperatively, the deviation does not usually change to any marked degree as a result of orthoptic training alone, unaided by the correction of ametropia.

TABLE 1

DEVIATION BEFORE OPERATION IN 324 PATIENTS

Deviation in prism diopters	Groups I and II No Prior Orthoptic Training		Group III With Orthoptic Training	
	No.	%	No.	%
20–40	81	37	45	47
41–60	146	63	52	53
Total	227	100	97	100

After operation the results were considered satisfactory from a cosmetic viewpoint if either orthophoria or heterophoria existed or the undercorrection was less than 15 prism diopters. In group I (no orthoptic training), after operation it was found that 22 percent of the patients were either orthophoric or heterophoric, while an additional 16 percent were undercorrected by 15 prism diopters; that is, 38 percent could be considered satisfactory from a cosmetic view-

TABLE 2

POSTOPERATIVE MUSCLE BALANCE

Muscle Balance	Group I No Orthoptic Training			Group II Postoperative Orthoptic Training			Group III Pre- and Postoperative Orthoptic Training		
	No.	%		No.	%		No.	%	
Orthophoria or Heterophoria	32	22	38	42	50	63	61	63	77
Undercorrection less than 15 prism diopters	23	16		11	13		14	14	
Undercorrection more than 15 diopters	73	50		22	27		16	17	
Overcorrection	16	12		8	10		6	6	
Total	144	100		83	100		97	100	

point. In group II, however (orthoptic training only after operation), 50 percent of the patients had either orthophoria or heterophoria, and another 13 percent had an undercorrection of less than 15 prism diopters. In group III (orthoptic training before and after operation) 63 percent were orthophoric or heterophoric while 14 percent were still undercorrected less than 15 prism diopters. Therefore, in this latter group, 77 percent had a good cosmetic result after operation (table 2).

In group I the amount of postoperative undercorrection was more than 15 prism diopters in 50 percent of the patients who received no orthoptic training, in 27 percent of the patients in group II following orthoptic training, and in only 17 percent of the patients in group III following preoperative and postoperative orthoptic training. Overcorrection was present in 6 percent of the patients in group III, 10 percent in group II, and 12 percent in group I; therefore, in our patients overcorrection occurred more frequently in the patients receiving no orthoptic training or only postoperative orthoptic treatment (table 2). However, the number of patients is too limited to permit sweeping conclusions to be drawn.

TABLE 3

RETINAL CORRESPONDENCE

	Group I				Group II				Group III			
	Pre-operative		Post-operative		Pre-operative		Post-operative		Pre-operative		Post-operative	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Normal retinal correspondence	61	42	64	45	30	36	65	79	73	75	80	82
Anomalous retinal correspondence	83	58	80	55	53	64	18	21	24	25	17	18
Total	144	100	144	100	83	100	83	100	97	100	97	100

RETINAL CORRESPONDENCE BEFORE AND AFTER OPERATION

Anomalous retinal correspondence was present in 61 percent of 227 patients (groups I and II) before surgery in the groups which received no preoperative training, while in group III, 75 percent of the patients had normal retinal correspondence after appropriate orthoptic treatment was instituted before surgery.

After surgery, anomalous retinal correspondence persisted in 55 percent of the cases in group I—a reduction of only 3 percent after surgical intervention (table 3). However, in groups II (79 percent) and III (82 percent) after surgery and orthoptic treatment, 81.5 percent of 180 patients had normal retinal correspondence (table 3)—an increase of 24.3 percent.

FUSION AND GRADE OF BINOCULAR VISION BEFORE AND AFTER OPERATION

Of 227 patients in groups I and II receiving no orthoptic treatment before operation, there were 62.3 percent who had no fusion preoperatively, while in group III (orthoptic training before and after operation), only 30 percent of the patients had no fusion preoperatively (table 4). Postoperatively, in group 1, 54 percent developed no fusion; that is, 9 percent of these patients apparently developed some degree of fusion, probably as a result of the operation. In group II, 20 percent of the patients developed no fusion after postoperative orthoptic treatment, while

in group III only 18 percent of the patients had no fusion on their maculae after preoperative and postoperative orthoptic training (table 4).

Of the 227 patients receiving no orthoptic training in groups I and II before operation, 11.5 percent had first-grade binocular vision preoperatively; 24.05 percent had second-grade binocular vision, and only 2.1 percent of the patients possessed third-grade binocular vision; whereas, in 97 patients receiving fusion training before operation, 35 percent developed first-grade binocular vision, 33 percent second-grade, and 2 percent third-grade fusion. After operation, of the 144 patients in group I (no orthoptic training), 12 percent had first-grade fusion, 27 percent had second-grade fusion, and 7 percent developed third-grade fusion. In group II after postoperative orthoptic training, 9 percent of the patients had first-grade fusion, 49 percent had second-grade fusion, and 22 percent had third-grade binocular vision. Of the 97 patients receiving preoperative fusion training, it was found that after operation and postoperative fusion training, only 13 percent remained with first-grade fusion, 38 percent developed second-grade fusion, and 31 percent had third-grade binocular vision (table 4).

Summary and Conclusions

An analysis of 324 cases of strabismus in which the patients were operated upon during 1938 to 1940 at the New York Eye and Ear Infirmary was made in order to determine the effect of preoperative and postoperative orthoptic training. The patients were divided into three groups. Group I (144 patients) received no orthoptic training; group II (83 patients) were given postoperative orthoptic training; while in group III (97 patients) preoperative and postoperative orthoptic training was administered.

TABLE 4

Fusion and grade of binocular vision

	Group I				Group II				Group III			
	Pre-operative		Post-operative		Pre-operative		Post-operative		Pre-operative		Post-operative	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
No Fusion	91	63	79	54	51	61.7	17	20	29	30	17	18
First-grade binocular vision	14	10	16	12	11	13	7	9	34	35	13	13
Second-grade binocular vision	34	24	39	27	20	24.1	40	49	32	33	37	38
Third-grade binocular vision	5	3	10	7	1	1.2	19	22	2	2	20	31
Total	144	100	144	100	83	100	83	100	97	100	97	100

Before operation a deviation of from 41 to 60 prism diopters was present in 63 percent of groups I and II (no preoperative orthoptic training) and in 53 percent of group III (after preoperative orthoptic training. Apparently the deviation does not usually change to any marked degree as the result of orthoptic training alone, unaided by the correction of ametropia.

Preoperatively abnormal retinal correspondence was present in 61 percent of 227 patients in groups I and II (groups receiving no orthoptic training before operation); by means of orthoptic training, normal retinal correspondence was established before operation in 75 percent of the patients in group III.

Before operation in group III (preoperative and postoperative orthoptic training), 35 percent of the patients had first-grade binocular vision and 33 percent had second-grade vision. In groups I and II (no preoperative orthoptic training) only 11.5 percent had first-grade binocular vision and 24.05 percent had second-grade binocular vision.

Following operation, orthophoria or heterophoria resulted in 22 percent of the patients in group I, in 50 percent in group II, and in 63 percent of the patients in group III; that is, the postoperative results were almost 300 percent better in the patients receiving orthoptic training before and after operation (group III) as compared with the group on which only surgery was performed (group I).

In our opinion orthoptic training is an indispensable adjunct to the surgical treatment of strabismus; that is, in overcoming anomalous retinal correspondence and amblyopia and in developing fusion with increased amplitude. Most of the actual reduction of the deviation in strabismus usually must be obtained by operation and the correction of ametropia.

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EXOPHTHALMOS

The first manifestation of thyrotoxicosis

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For the ophthalmologist to be the first in routine ocular examinations to recognize diabetes, hypertension, Bright's disease, or adenoma of the pituitary is not an uncommon occurrence. However, to be the first physician to diagnose thyrotoxicosis because of an ocular complaint is indeed unusual and uncommon.

Exophthalmos is a unilateral or bilateral protrusion of the eyeball in its bony socket. There are many causes for its manifestation, but at this time we are concerned only with its relation to Graves's disease. The term exophthalmos is frequently used loosely and confused with Dalrymple's sign, which is a retraction of the upper lid simulating a protruded eyeball. Hertel's exophthalmometer makes its measurement possible and thus confusion may be avoided.

Unilateral exophthalmos is not of necessity etiologically similar to the bilateral condition. Frequently many conditions of the unilateral state may be simply incomplete in terms of bilateral involvement. Of this, exophthalmic goiter is perhaps the outstanding example.

Proptosis constitutes one of the most conspicuous and important signs of this disease and is present in about 75 percent of the cases. It occurs in its prominent form in the young. Late in the disease, and particularly after thyroidectomy and a low basal metabolic rate, it may present one of its worst phases in the form of malignant exophthalmos.

Discussion with many of the physicians well versed and familiar with the eccentricities of thyroid disease has elicited the fact that exophthalmos as the first sign, preceding clinical and laboratory manifestations, is unusual, whereas the presenting form is not uncommon. Some of these clinicians interrogated could vaguely recall having seen one or two isolated cases but could not substantiate these thoughts with specific cases or clinical data. However, there was a concurrence of opinion as to its rarity.

E. C. Ellett[1] cited two cases of Graves's disease in which the exophthalmos preceded the signs and symptoms of thyrotoxicosis. In one patient, a woman aged 23 years, the interval was one year. In the other, a man aged 27 years, it was two years. Unfortunately, in the latter instance a diagnosis of tumor of the orbit was made and an exploratory operation performed. No tumor was found. After describing these two cases Ellett states that proptosis preceding thyroid disturbance is "not an unusual condition." Such, however, has not been our experience, and the paucity of literature on the subject and its rarity prompts this discussion and the presentation of three cases.

Etiology

Opinion as to the causation of exophthalmos is still divided. For many years the popular theory that it was the result of excessive thyroid secretion was accepted by most. In a short time the literature contained many cases in which malignant exophthalmos occurred in the face of diminished thyroid function, especially following thyroidectomy. In fact, this surgical procedure seemed to favor its development in many cases. Consequently this hypothesis as to origin soon became difficult to accept. If at all, the thyroid gland must play a secondary role

in its production, for exophthalmos may be experimentally induced much more easily following thyroidectomy by means of thyrotropic hormone. We are also familiar with the therapeutic use of desiccated thyroid as a means of alleviation after operation has failed to retard its progress. In all probability we may postulate that it is the result of polyglandular disturbance and imbalance.

Pathology

The mechanism and pathology of exophthalmos has excited many discussions in the past. There were adherents to the hypothesis that retrobulbar venous engorgement was the cause, and that the engorgement was brought about by a vasomotor toxin. The hypothesis of protrusion of the globe by dilated orbital arteries had numerous proponents, but this theory lost its popularity. It was also explained on the basis of a weakening and lengthening of the extraocular muscles, and this too fell into disrepute.

Subsequently the smooth periorbital muscle was indicated as the offender. It was alleged that its stimulation caused the orbital contents to protrude. In the lower animals this muscle is proportionately large; in man it is vestigial. Because of this fact, the theory soon became untenable.

Another explanation suggested an increase in the orbital fat as the mechanical factor. The weakness of this hypothesis lay in the known fact that thyrotoxicosis causes an early loss of body fat.

It remained for Naffziger[2] and his group to settle this pathological discussion in 1931. Grossly, he found the extraocular muscles to be enlarged three to eight times in diameter.[3] Histologically, various stages of degeneration were observed, with interstitial edema, proliferation of small round cells that tended to accumulate about blood vessels, and finally, fibrosis, which appeared more dense and extensive in the more advanced cases.

Experimental exophthalmos

Since 1931 it has been known that exophthalmos in animals can be produced by the injection of anterior-pituitary extract. In 1932 Loeb and Friedman[4] induced proptosis in guinea pigs with injections of the acid extract of anterior pituitary of cattle. The following year, Marine and Rosen[5] induced it in guinea pigs by the intraperitoneal injections of the same extract. They further demonstrated that it occurred readily in thyroidectomized guinea pigs. In 1932, Friedgood[6] with daily injections produced it on the tenth day when the basal metabolic rate was highest and the thyrotoxicosis most marked. It was further disclosed that the exophthalmos became very marked if the injections were continued over a period of months, especially in animals which presented a low metabolic rate.

The aforementioned experiments have since been repeated by many investigators. Aird[7] went one step further, and in an analysis of his anterior-pituitary extract ruled out the gonadotropic, mammotropic, and growth hormones as factors in the production of proptosis. He reached the conclusion that the thyrotropic principle was responsible for the exophthalmos and associated extraocular myopathy. The changes in the extraocular muscles of the experimental animals were similar to those found in humans afflicted with exophthalmic goiter. Thus, it is suggested that the thyrotropic hormone is the factor of fundamental importance and that the myopathy is the end result of an irreversible malignant exophthalmos.

Cases

Case 1. C. H., a man, 32 years old, married, in June, 1938, had noted a "stary

right eye" for the past two weeks. His vision was normal, and he had no other physical complaints. Past history, family history, and marital history were irrelevant.

External examination disclosed an exophthalmos of the right eye of 2 mm., as measured with Hertel's exophthalmometer. There was no lid lag, and muscle excursions in all directions were equal and not limited. The red-glass test showed no diplopia. The pupils were equal and reacted normally to light and accommodation. Tension and media were normal. The eye grounds presented normal discs; no abnormalities of the vascular tree, exudates, nor hemorrhages were seen. On palpation the right eye offered no resistance to its return into the orbit. No masses nor tumor could be felt around the orbital margin. No pulsation could be felt nor any bruit heard. X-ray studies of skull and orbit were negative.

A complete physical examination at this time elicited no evidence of thyroid disease. The thyroid gland was normal. The patient did not show weight loss, palpitation, excessive perspiration, nervousness, or fine tremors of the fingers. As a result of all these negative findings a tentative diagnosis of psuedo-tumor of the right orbit was made.

The patient was seen again in October, 1938, at which time no change in the foregoing findings was obtained except that Hertel's exophthalmometer disclosed 3 mm. of exophthalmos. He volunteered the information that the proptosis became worse with fatigue and was slightly less when he awakened in the morning, but never entirely disappeared.

The condition remained unchanged until June, 1939, when the patient became ill with an epidemic parotitis which kept him in bed for two weeks. During this time, he lost five pounds and failed to regain it, although his appetite had increased. Again the thyroid gland was normal; none of the cardinal signs of thyrotoxicosis were visible.

In January, 1940, he became ill with la grippe and lost eight more pounds. Following this illness he noted that his legs trembled and felt shaky. He was weak and became easily fatigued. He became nervous, and in spite of an increasing appetite failed to regain his weight.

Examination at this time disclosed 4 mm. of exophthalmos and lid lag in the right eye. The thyroid gland presented a right lobe twice normal in size with the left lobe slightly enlarged. The rest of the examination was negative except for tremors of the fingers. Basal metabolic rate was +16 and when repeated in four days was +13. Blood chemistry and urine analysis were negative. It was at this time that a diagnosis of Graves's disease was made.

In March, 1940, under local anesthesia a bilateral subtotal thyroidectomy was performed and the patient made an uneventful recovery. Pathological report on the specimen stated "moderate hyperplasia and involution of the thyroid."

Thus, the time interval between the onset of exophthalmos and the symptoms of thyrotoxicosis was one year and eight months.

The exophthalmos receded somewhat after operation but has never entirely disappeared; it is still aggravated by fatigue.

Case 2. R. G., a single woman, aged 20 years, was seen in August, 1935. She stated that she had been well, but her friend called her attention to her "stary eyes." No abnormalities of vision were noted, and the patient had no physical complaints. Past history and family history were irrelevant. External examination presented bilateral exophthalmos. No pulsation could be felt nor bruit heard with the stethoscope. There was no lid lag, and muscle excursions were equal

and normal in all cardinal directions. The red-glass test disclosed no diplopia. No masses nor tumor could be felt at the orbital margins. The pupils reacted normally to light and accommodation. Tension and media were normal. The eyegrounds disclosed normal discs, normal retinal vascular tree, and no hemorrhages nor exudates.

Physical examination was completely negative. The thyroid gland showed no enlargement. There was no loss of weight, palpitation, excessive perspiration, nervousness, nor tremors of the fingers. X-ray studies of orbits and skull were negative.

During the next five months the patient's condition remained the same. In December, 1935, her eyes had returned to normal, and a short time after this she was married. Three weeks later she became weak, suffered from cramps, diarrhea, and loss of weight, symptoms which she attributed to the excitement of matrimony. Her exophthalmos had not only returned but had increased. At this time two successive metabolisms were done and disclosed +25 and +35, respectively.

Complete blood examination was negative except for a blood cholesterol of 111 mg. per 100 c.c. Urine analysis was negative.

In January, 1936, under local anesthesia a subtotal bilateral thyroidectomy was performed. Much of the gland was found to be substernal. The patient made an uneventful recovery.

Pathological report of the specimen stated "hyperplastic thyroid with involution changes."

In this case the time interval between the onset of exophthalmos and thyrotoxicosis was six months. After operation, both eyes receded to normal positions.

The next case is of more than passing interest in that it opens many avenues of consideration in the field of heredity in thyroid disease. This last patient is a younger brother of the first patient.

Case 3. P. H., 30 years old, married, was seen in May, 1940, with the complaint of a glassy appearance of the right eye for the past month. The vision was normal and the patient had no other physical complaints. His past history and marital history were irrelevant.

External examination presented an exophthalmos of 1½ mm., as measured with Hertel's exophthalmometer. There was no lid lag, and muscle excursions in all directions were equal and not limited. The red-glass test disclosed no diplopia. Pupils were equal and reacted normally to light and accommodation. Tension and media were normal in each eye. Eye grounds disclosed normal discs, no abnormalities of the vascular retinal tree, no exudates, and no hemorrhages. On palpation the right eye offered no resistance to its return to the orbit. No masses nor tumor could be felt at the orbital margin. No pulsation could be elicited nor bruit heard with the stethoscope. X-ray films of skull and orbits were negative.

Complete physical examination at this time presented no evidence of thyroid disease. The gland was normal to palpation. There was no loss of weight, palpitation, excessive perspiration, nervousness, nor tremors of the fingers. The basal metabolic rate was +8.

In September, 1940, with Hertel's exophthalmometer the exophthalmos measured 2 mm. in the right eye. The patient had gained in weight and presented no signs nor symptoms of thyroid disease.

A therapeutic test with iodine was made for 13 days, with no change in the proptosis or constitutional condition.

In November, 1940, he presented 2½ mm. of exophthalmos. There had occurred no loss in weight and none of the usual signs nor symptoms of thyroid disease were present.

In December, 1940, his exophthalmos measured 3 mm. in the right eye. There occurred no change in his physical condi-

tion. However, he volunteered the information that usually he was extremely sensitive to the cold weather but that of late the coldest days caused him no discomfort; in fact, he always felt warm.

In January, 1941, the patient presented a 3-mm. exophthalmos in the right eye. He had lost five pounds in weight in spite of a good appetite. He perspired more easily. Physical examination disclosed the gland normal to palpation. The fingers manifested a fine tremor. A diagnosis of Graves's disease was made and temporarily he is under medical care.

The interval between exophthalmos and the onset of clinically manifest thyrotoxicosis was eight months.

Comment

Exophthalmos as an accompanying sign of thyrotoxicosis is very common and frequently may be aggravated by thyroidectomy. As the first and earliest sign of thyroid disease, it is uncommon. Assuming the theory that the thyrotropic hormone of the anterior-pituitary gland is the primary and fundamental factor in its production, it is difficult to explain why this hormone acts on the extraocular muscles first and a subsequent date produces the classical signs and symptoms of exophthalmic goiter. It is also difficult to explain why the extraocular muscles should be selected and no other voluntary muscles of the body affected. There have been isolated cases in which other muscles have been attacked, but these are extremely rare.

Aware of the fact that toxic thyroid cases may spontaneously enter a phase of remission, it may be suggested that the foregoing cases fall into such a clinical state, but it is still difficult to explain the continued action of the hormone on the extraocular muscles. It is possible that these patients may be mild cases of Graves's disease undetectable by present methods of examination. In addition, it is suggested that the therapeutic test with iodine should be used as a possible means for the detection of such early and mild examples of hyperthyroidism.[8]

Although it is postulated that the thyrotropic hormone is responsible for both hyperthyroidism and exophthalmos, in humans with this syndrome we find no evidence of the thyrotropic hormone in the blood. Is it conceivable that the two conditions are produced by two independent factors? May the possibility be presented that the exophthalmos is produced by the thyrotropic hormone and the thyrotoxicosis by neurogenic factors? This problem opens a fertile field for further investigation.

Summary and Conclusions

Three cases are presented and described in which the first or only complaint was exophthalmos. The condition progressed in spite of continued negative signs and symptoms. The diagnosis of Graves's disease was made in each case after an interval of 20 months, 6 months, and 8 months, respectively.

An interesting sidelight in this presentation was the fact that two of the patients were brothers, which suggests the importance of heredity in Graves's disease.

In the differential diagnosis of exophthalmos, when the physical, chemical, and X-ray examinations and determinations are negative, exploratory operation of the orbit for tumor should be delayed until Graves's disease is definitely excluded.

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NOTES, CASES, INSTRUMENTS

RETINOCHOROIDITIS OF THE JENSEN TYPE WITH SECONDARY GLAUCOMA*

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The following case of choroiditis is considered to be of interest because of some unusual features, including the presence of a high intraocular tension, resemblance to an intraocular tumor and the occurrence of a sectorlike defect in the field.

The situation of the lesion away from the disc places it outside the category of retinitis juxtapapillaris of Jensen. In other particulars of morphology and clinical course it follows so closely the previous descriptions of Jensen's disease that I have considered it to be a retinochoroiditis of the "Jensen type," and as such it falls definitely, I believe, in the group called by Heath[1] "Solitary retinochoroiditis."

Miss S. L., 34 years of age, first consulted Dr. Joseph Keiserman on November 20, 1939, complaining that for some months there had been increased prominence of the right eye. About one week before this consultation there had been a period of rather severe pain in the right eye. This had been followed by an inconstant floating spot in the upper field, and also by what was described as a "dark line" in the lower field.

A complete and careful physical examination by Dr. Keiserman revealed no positive findings except tonsillar stumps, a systolic mitral murmur and increased intraocular tension to palpation in the right eye, for which he referred the patient for diagnosis and treatment.

The previous medical history included the removal of a reddish tumor (designated a hemangioma) from the anterior chest wall by "burning" in December, 1938, with no local recurrence. There had also been one severe attack of paroxysmal tachycardia, recurrent extra systoles, and one attack of pain in the joints and muscles during September, 1938. Blood counts were normal. The patient was of the nervous and apprehensive type and quite intelligent.

Fig. 1 (Cowan). Ophthalmoscopic appearance of fundus of right eye on November 21, 1939.

Ocular examination, November 21, 1939, showed no undue prominence of either eye. In the right eye there was a slight corneal haze, and its pupil was slightly larger. Both pupils reacted to light and accommodation normally. There was no conjunctival injection. The anterior chamber was of normal depth. The slitlamp showed only a slight bedewing of the corneal epithelium in the right eye. The visual acuity in both eyes was 6/6+. Intraocular tension was 48 mm. in the right and 20 mm. Hg in the left eye (Schiötz).

* Read before the Section on Ophthalmology of the College of Physicians of Philadelphia, February 15, 1940.

The ophthalmoscopic examination of the right eye showed a slight corneal haze but otherwise clear media. The disc margin was slightly blurred above, otherwise normal. About one and one-half disc diameters above and slightly lateral to the

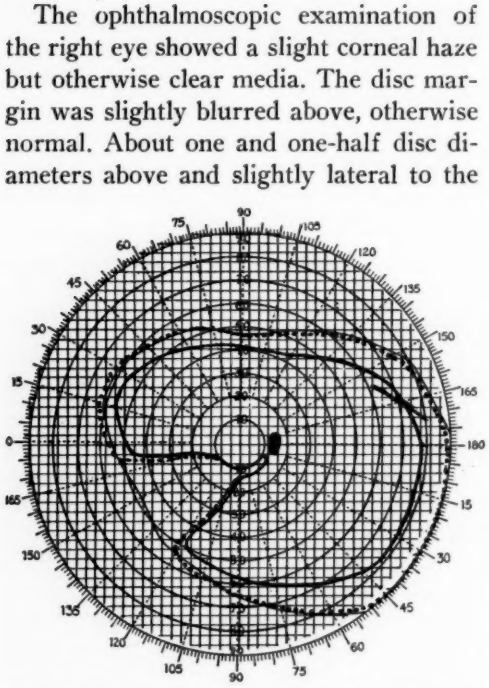

Fig. 2 (Cowan). Visual field on December 12, 1939. Solid line = 5/1000; dotted line = 15/1000.

disc was a grayish-white, rounded lesion with rather well-defined margins, smooth contours, slightly raised above the retinal level (fig. 1). Despite the lack of pigment in the lesion, its appearance, together with the increased tension and the history of the removal of a tumor from the chest, brought up for consideration the possibility of an intraocular neoplasm. Acute choroiditis was, of course, also considered, but no vitreous opacities or K.P. was seen, and the smooth, solid, raised appearance and sharp outline made this diagnosis uncertain. The fundus of the left eye was normal.

A strong miotic mixture (pilocarpine nitrate, gr. i; eserine salicylate, gr. 0.5; aqua, dr. ii) was prescribed, together with hot compresses. This was used for 6 days, and the patient was observed daily, with no variation in the appearance of the lesion. The intraocular tension in the right eye ranged from 30 mm. to 70 mm. Hg; the tension in the left eye remained normal at all times. During this period an X-ray study of the teeth was made and one premolar, with a granuloma at the root, was extracted.

On November 26, 1939, for the first time, a number of fine translucent deposits on the posterior surface of the cornea were seen. Many fine vitreous opacities were also present, which however, only slightly obscured the fundal view. The retinal lesion now presented less distinct margins and a more fluffy texture. The disc margin above was well defined. The visual acuity was 6/12 and the intraocular tension 72 mm. Hg at this time. The entire clinical appearance was definitely that of an acute exudative choroiditis of a mild type, aside from the high tension. The miotic mixture was now stopped, atropine, one percent, was instilled daily and one-percent dionin ordered. The patient was seen November 27, 1939, by Dr. W. I. Lillie, who concurred in the diagnosis of posterior uveitis.

Fig. 3 (Cowan). Fundus of right eye on January 3, 1940.

Under this regimen the tension returned to normal the next day. The retinal lesion began to recede and diminish in

size, the vitreous opacities to absorb, and the vision to improve. On December 12, 1939, the lesion was much smaller, the vision 6/6+ in both eyes. A few fine vitreous opacities and precipitates on the posterior cornea were present. A field, taken at this time to determine the effects of the continued high tension, showed a large sectorlike cut which ran from a point near the blind spot corresponding to the site of the lesion, to the lower nasal periphery (fig. 2). The defect was absolute with very steep borders.

Three weeks later examination showed the lesion illustrated by figure 3. This consisted of a yellowish gray, slightly depressed scar, just under the vein over which the exudative mass had previously lain, and above the vein an area of fine pigment stippling. The whole was surrounded by lighter aureole of retinal depigmentation. The artery near the lesion showed some irregularity of caliber and increased reflexes.

One year later the appearance of the lesion was the same, except that the arterial caliber was less irregular. Little or no increase in pigmentation had taken place. The field was identical with that taken previously. Vision remained 6/6+ in both eyes and the intraocular tension was normal. The patient's general health was reported as excellent.

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A SIMPLIFIED ENTOPTIC PUPILLOMETER

DAVID G. COGAN, M.D.
Boston

Entoptic pupillometry is the most accurate and practical method for measuring the size of the pupil in human beings. Yet it is a method that is little used, probably owing to the complexity and inaccessibility of the apparatuses that have been previously employed. I am led, therefore, to describe a simple entoptic device that I have found entirely satisfactory. It may be made in 5 minutes' time, with no more equipment than a pin, a piece of cardboard, and a millimeter rule. It is suitable for the clinic or for the laboratory wherever subjective measurements are to be made and, in my opinion, is as sensitive as any of the more elaborate methods heretofore reported.

Entopic visualization ordinarily consists in the perception of a shadow on the retina from some intraocular structure. It is present only when the light source is not focused on the retina. The pupil is seen entoptically as a luminous field bounded by the shadow of the iris. The nearer the light source is to the eye, the bigger the pupil will appear. If the light source is at the anterior focal point the luminous field projected on the retina will approximate the actual size of the pupil. Inasmuch as it is impracticable to have the actual light source as close to the eye as is desirable for entoptoscopy, an effective light source may be substituted either by using a pinhole held in front of the eye in conjunction with a luminous background such as the sky, or a small reflecting surface, such as a shiny brass knob, with the actual light to one side and behind the observer.

It is possible within the limits of the eye's revolving power to visualize entoptically as many pupils as there are effective light sources. In entoptic pupillometry single or double pupils are used. The single pupil is employed in the pupillome-

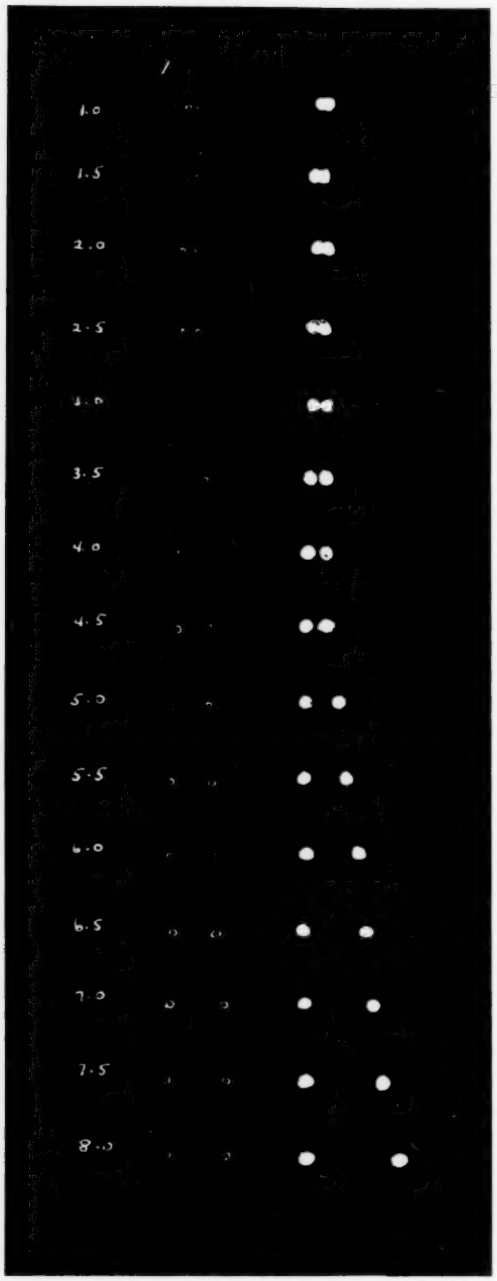

Fig. 1. (Cogan). Simple entoptic pupillometer.

ters of Lampis,[1] Viallefont and Lafon,[2] and Jones.[3] Here the effective light source must coincide with or be at a fixed distance in front or behind the anterior focal point. The luminous field corresponding to the pupil is projected on to a grating. The double pupil is employed in the pupillometers of Broca,[4] Badal[5], Houdin[6], and by Moss.[7] Here the distance between the two effective light sources is varied, until the two entoptic pupils appear tangential to each other. The distance between them is then the diameter of the pupil. With the double pupils the effective light sources may be anywhere within the punctum proximun, although it is most convenient to have them close to the eye. The apparatuses heretofore described for both types of pupillometry are relatively elaborate.

The apparatus and technique that I have employed are as follows: Through a piece of cardboard or fiber board a series of paired holes is made such that the separation of the holes in any one pair is 0.5 mm. greater than that of the preceding pair. The pairs are arranged in a vertical column starting with a separation of 1 mm. and ending with a separation of 8 mm. The distances apart of the successive pairs is immaterial. I have found 1 cm. satisfactory. I have found it convenient to illustrate beforehand what the patient should see entoptically by drawing sample circles on the pupillometer adjacent to the holes (fig. 1). On the other side of the holes is the measure of their distance apart.

The patient is instructed to hold the card close to his eye and look at the sky or lighted wall through any pair of holes. He is then asked to raise or lower the card until he finds that pair which produces luminous discs that are tangential to each other. The separation of the holes in this pair will then be the diameter of the pupil. The sensitivity is well within 0.5 mm., and spontaneous fluctuations of the pupil make any greater accuracy unnecessary for ordinary purposes. Unless

consensual reactions are being measured the eye that is not being tested should be occluded.

The advantages of this pupillometer, aside from its simplicity, are the ease and the rapidity with which an untrained person can make the measurements. It has the further advantages over all objective tests, with the possible exception of photography, that the amount of light entering the eye is insignificantly small and that movements of the eye during the measurement do not interefere with the test. I have even employed it successfully in the presence of nystagmus.

243 Charles Street.

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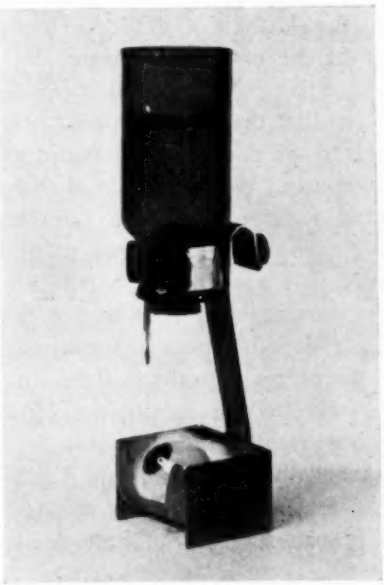
APPARATUS FACILITATING STUDY BY BIOMICROSCOPY*

Parker Heath, M.D.
Detroit, Michigan

This device consists of a rustless trough eye holder above which is an inverted bottomless bottle. The cornea of the animal eye (rabbit, cat, dog, pig, calf) is kept moist by physiologic salt solution dropped through a grooved cork supporting a wick. The design is simple and facilitates study of the animal eyes. This enables the student to practice technique in biomicroscopy leisurely. There is a fastening device on the back. It is made by the White Haines Optical Company, Detroit, Michigan.



* From the Department of Ophthalmology, Wayne University.

SOCIETY PROCEEDINGS

EDITED BY DR. RALPH H. MILLER

COLLEGE OF PHYSICIANS OF PHILADELPHIA

SECTION ON OPHTHALMOLOGY

October 17, 1940

DR. FRANCIS HEED ADLER, *chairman*

PNEUMOCOCCUS INFECTION OF THE CONJUNCTIVA FOLLOWED BY A PNEUMOCOCCUS MENINGITIS

DR. CARROLL R. MULLEN AND DR. JOSEPH R. WEINTROB reported on this case.

In the death of a child due to meningitis of pneumococcic origin, it is of more striking ophthalmologic interest to observe that the first complaint and sign appeared in a pneumococcus conjunctivitis of one eye. While this local condition cleared rapidly and quite satisfactorily under treatment, the signs pointing to meningitis began while the child was still under hospitalization. It is not possible to state definitely that the conjunctivitis was the origin or source of the infection which resulted in meningitis. However, it is important to note that this child was in apparently good health until the onset of the conjunctivitis, which was followed in a few days by the fatal attack of meningitis.

This case is reported from the ophthalmologic service of Dr. Carroll R. Mullen and the pediatric service of Dr. Ernest L. Noone at the Philadelphia General Hospital: The patient was a one-month-old male infant. On September 19, 1940, the child developed a redness in the right eye which was treated at home with boric acid. On the following morning the patient was admitted to the Philadelphia General Hospital with a markedly inflamed and swollen right eye. The lids were thickened and of an angry red color. The conjunctival sac was bathed in pus. A smear showed many gram-positive diplococci resembling pneumococci. Otherwise the child was entirely well, showing no evidence whatever of meningitis or other disorder. The patient was placed on sulfathiazole solution as well as 0.25-percent atropine sulphate, three times a day, and frequent cold compresses.

In 48 hours the eye was entirely normal and all treatment was stopped except irrigations every three hours. The child appeared to be entirely normal. During the following 24 hours, the child's temperature rose to 103°F., and he began to have frequent convulsions. The tentative diagnosis of meningitis was made, and this was borne out by the examination of the spinal fluid, which revealed large numbers of pneumococci, type V, as well as many pus cells.

Despite three blood transfusions and large doses of type-V antipneumococci serum and sulfapyridine by mouth in adequate doses, the condition became progressively worse and terminated in death. Post-mortem examination confirmed the diagnosis, having shown a plastic purulent meningeal exudate which on culture contained a pneumococcus, type V. Post-mortem did not reveal a course or mode of extension from the eye, if such was the focal point.

Discussion. Dr. Ernest L. Noone said that the question of primary importance in this case was whether the conjunctivitis was the original source of infection and the direct cause of the meningitis, or whether a hidden infection of the post-

nasal space, possibly with invasion of the infantile ethmoid, initiated the conjunctivitis and meningitis. He could not answer that question. When he first saw the case, the conjunctivitis had been cleared up and the eye was in excellent condition. There was mucus and pus in the postnasal space, but that might have come from a later secondary infection.

It is difficult to know the origin of infection from this area. This is an intricate maze and he was not anatomist enough to trace the possible avenues. Infection may travel by way of the angular vein to the cerebral sinuses, but it may also travel directly from the ethmoids into the longitudinal and cavernous sinuses. The autopsy report indicated that the sphenoid was red and spongy, but this is the usual appearance in infants of that age. Dr. Helen Riggs had told him that the thrombus described in the longitudinal sinus might be discounted as the primary source of meningeal infection. Had that been the case there would have been more destruction of the surrounding tissue. It was probably a terminal event secondary to circulatory failure.

Type-V pneumococcus is found more frequently in children over two years of age than in infants of one month. The patient was treated with rabbit serum type V and with sulfapyridine. Previously he had been given sulfathiazole. Sulfathiazole may have had a deterrent effect upon any postnasal infection or even upon an arising meningitis. This drug is not very effective in the treatment of meningitis because of the low concentrations in the spinal fluid; the effective concentration has not yet been reported, within the speaker's knowledge.

Dr. Noone questioned the wisdom of the two cisternal punctures performed on the last day of the infant's life. In a baby five weeks old the cisterna is a very small space and he thought that ventricular puncture might be safer. However, the punctures in this case, in his opinion, had nothing to do with the fatal outcome. Specific serum and chemotherapy are now available to build up adequate concentration in the spinal fluid.

Dr. Carroll R. Mullen said that the usual source of meningeal infections comes by way of the middle ear. In this patient the nasopharynx was explored and no involvement could be found in the original examination. He had never known a case in which a primary conjunctival infection had resulted in an attack of meningitis. Reports of patients have been made in which meningitis followed enucleation of the globe in panophthalmitis, but Gradle has pointed out that autopsies on such cases have never revealed a direct source of continuity of the infection. It must therefore travel back through the blood stream; which could have happened in the present case. He did not insist that the conjunctival infection was the original source. The child had been given a careful examination in the pediatric out-patient department, and all unusual signs were looked for. Sulfathiazole had been given immediately upon admission, locally and by mouth. Meningitis was not suspected. Dr. Mullen said he had been able to observe the effect of sulfapyridine in cases of pneumococcic infection of the conjunctiva and in one such case the patient was cured within 24 hours.

Dr. Warren S. Reese. Were bacteriologic studies of the nose and throat made?

Dr. Mullen: No, none were made.

Dr. Weintrob, closing, in answer to Dr. Block's query of how to account for the high temperature when the condition was considered purely localized, said that this was a rectal temperature and above the normal 99.6 degrees, about 0.6 to 0.7 of a degree, and therefore aroused no suspicion.

Microphthalmus with tumorlike neuroglial proliferation

Dr. W. E. Fry presented a report concerning a microphthalmic eye which demonstrated marked intraocular changes, but which was not associated with a cyst, either by clinical or histological examination. Dr. Shipman had enucleated the eye and permitted the report.

The measurements of the eye were well below those of a normal eye at birth: antero-posterior diameter, 8 mm.; lateral, 8½ mm. Normal minimal dimensions at birth would be 12.5 and 14.5, respectively. The cornea measured 7.5 mm.; normally it would measure 10 mm.

Without describing the eye in detail the following facts should be pointed out: The choroid appeared to be normally formed and the corneal stroma and sclera well developed. There was no lens and no lamina cribrosa. The hyaloid artery was present. The angle of the anterior chamber was poorly developed and appeared similar to illustrations of the angle of a five-month fetus. No normal iris tissue was present but a band of tissue separated the aqueous from the vitreous chamber. This band consisted of collagenous tissue on the posterior aspect. The pigment epithelial layer of the retina was present. The whole of the vitreous cavity contained a mass of tissue that was partly collagenous tissue surrounding the hyaloid artery and partly proliferated neuroglial tissue. Groups of cells similar to those which made up the internal nuclear layer were present. There was no separation into internal and external nuclear layers. There were no rosettes.

The marked change in the posterior part of the eye appeared to represent defective development of the inner layer of the secondary optic vesicle with a secondary proliferation of neuroglial tissue. Velhagen reported a somewhat similar case, but with less extensive neuroglial proliferation. The lack of a lens represented defective development of the surface ectoderm. The frequency with which cataract is associated with microphthalmus indicates that defective development of the associated ectoderm must be common.

Discussion. Dr. James Shipman said he would like to express his gratitude to Dr. Fry for bringing this case to his attention. Clinically, the child presented a typical picture of a microphthalmic eye which was somewhat irritable. It was removed for cosmetic reasons, and a gold ball was inserted in Tenon's capsule, a Frost-Lang gold-ball implant. The idea was to help the development of the orbital socket, as the child grew older. So far, the gold ball has stayed in place, and it is hoped that the operation has not been done in vain from this point of view. Dr. Shipman was surprised at the size of the cornea. It looked larger than it did grossly. The pathological findings as demonstrated were totally unknown to him and were not suspected. It is a revelation to see what is sometimes found in an eye.

Dr. Perce De Long said that anomalies of this type result from the faulty development of the primary optic vesicle. They may be divided into two groups, anophthalmus and microphthalmus. In the group known as anophthalmus the primary optic vesicle has not developed out of the cerebral vesicle. There is also in this group misdirected or faulty development of the primary optic vesicle, which leads to the development of cyclopia, and sometimes to the development of diprosopia, or two facedness. Anophthalmus can be distinguished from microphthalmus by the fact that in anophthalmus there are never any neurologic or nervous elements. Microphthalmus is distinctive as a small eye or a mal-development in which

neurologic elements are present. Now, in these two, as Dr. Fry has shown, there are two types again: the type in which the ectodermal layer has not been pushed back, in which we get the microphthalmus known as the cystic type. When the ectodermal layers are involuted, and thus become adjacent to the pigment layer of the choroid or the retina, they belong to the second group, in which this case belongs, for here it will be noted there is a sort of false retinal detachment which extends forward to the iris. The so-called glial tissue is the result of some insult. The surprising part of this section is the fact that usually in this type of deformity there are associated colobomas of the iris, lens, and choroid (as Dr. Fry said) but none were present in this specimen.

Dr. Francis Heed Adler asked how often the condition occurred. He thought there was something in Dr. Fry's mind of the possibility that this tumor had something to do with a retinal blastoma. He was looking for rosettes of Weinsteimer and they were not found. He asked whether it might be a glial placement, a real tumor, or a retinal blastoma.

Dr. W. E. Fry replied that he did not believe that this represented any real tumor formation. Neuroglial hypertrophy in nervous tissue corresponded to fibrous-tissue hypertrophy elsewhere, just as a keloid or a fibroma might be considered a tumor. He did not believe that it was a malignant growth. It was not a neuroblastoma since the neuro-epithelial characteristics were absent.

Surgical treatment of neurofibromatosis (plexiform neurofibromata)

Dr. Edmund B. Spaeth presented a paper on this subject in which he stated that plexiform neurofibromatosis, a subdivision of those cases grouped with von Recklinghausen's disease and neuromata, is surgically a neurofibroma of the terminal fan of the superficial nerve. He presented 11 such cases to illustrate the various defects which need correction, and described the surgical procedures necessary to achieve this.

Discussion. Dr. Isaac Tassman expressed his thanks to Dr. Spaeth for his very fine presentation and inquired what was the period of time in which he collected this unusually large number of cases of neurofibromatosis. He thought that it was a rather rare condition, and since one patient did show a pigmentation around the region of the knee joints, he asked whether Dr. Spaeth had determined in any other cases if tumor formations were present in any other part of the body. He also asked whether the eye grounds were examined and whether a report of the findings in the good eyes had been made.

Dr. Edmund B. Spaeth in closing said that all these cases were typical of plexiform neurofibromata, and showed no other tumor formations anywhere, but perhaps 50 percent of them showed cafe-au-lait spots. In all instances the opposite eye was normal. The collection of these cases extended over a period of 20 years.

Warren S. Reese,
Clerk.

SAINT LOUIS OPHTHALMIC SOCIETY

October 25, 1940

Dr. J. F. Hardesty, *president*

Experimental and clinical use of choline in relation to vascular disease

Dr. Wendell L. Griffith and Dr. G. O. Broun presented a paper on the above subject.

Discussion. Dr. W. H. Luedde said that none of the cases reported had been un-

der observation more than six or eight months. He suggested the improvement shown might be only temporary. He suggested also that the real value of the drug would be proved only by more widespread usage and the study of further reports.

Abduction and adduction ratio and its relation to refraction

Dr. M. L. Green presented the above communication.

Discussion. Dr. J. F. Hardesty stated that he believed most ophthalmologists at times saw patients who had an overcorrection or an undercorrection of their refractive error. He believed one should investigate before any criticism was made to make sure that the prescriber had a reason for ordering the correction for the patient. It has been found by some refractionists to be very efficacious in giving the patient relief. He stated that Dr. Green has been struck by the fact that patients' adductive and convergent powers are weaker at the end of the day than in the morning. When he examined applicants for the air corps in the afternoon and found that they did not have enough abductive power he had them come back in the morning for another examination.

Proliferating retinosis

Dr. William H. Meinberg presented two case reports.

Discussion. Dr. R. E. Mason said that the cases of retinitis proliferans that have come to his attention were those that followed injuries to the vitreous chamber from penetrating foreign bodies. He found that patients suffering an injury in the vitreous chamber from a foreign body or penetrating wound do not get an endophthalmitis so frequently since the advent of foreign-protein therapy, but a striking number of patients lose a part or all of the vision from a retinitis proliferans. He has become very cautious in giving a prognosis for a period of 10 days to three weeks following the injury, in cases in which a hemorrhage has occurred in the vitreous chamber. Connective tissue, proliferation strands, detachments, and organized exudates followed by part or great loss of vision frequently result. He wondered how a person could plunge a Graefe knife into the vitreous chamber with a certain amount of safety while a penetrating wound from a foreign body frequently induced a retinitis proliferans. He suggested that the explanation might lie in the production of a low-grade infection with a foreign body while none was introduced with a clean Graefe knife. He said he had been particularly embarrassed on making a prognosis on a permanent loss or partial loss. Frequently, after deciding an injured eye had cleared up, a strand of connective tissue pulled the retina loose, causing a great loss of vision. He thought it advisable for a patient who had a penetrating wound of the vitreous chamber to remain quiet for a period of two or three weeks and be treated with the same respect as a cataract patient.

Adolph C. Lange,
Editor.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

October 16, 1940

Dr. Morris Davidson, *presiding*

Visual requirements for army, navy, and aviation

Dr. Frederick Theodore read a paper on this subject during the instructional hour.

War injuries involving penetration of the eye and orbit

Dr. Allen Greenwood of Boston discussed his experiences in France during the First World War. He pointed out the

importance of ophthalmology in skull wounds. Furrow wounds often produced brain damage with consequent changes in intracranial pressure, as shown by the choking of the disc. This required immediate trephining of the skull. With the ophthalmoscope it was often possible to distinguish between choking of the disc due to increased intracranial pressure and optic neuritis due to meningitis. This was important in the determination of the immediate procedure to be adopted at the field hospitals.

Injuries of the eye and orbit from shrapnel, if at all extensive, required immediate enucleation or evisceration to prevent sympathetic ophthalmia. The wounded man's wishes were not consulted. During all his time in France Dr. Greenwood saw only one case of sympathetic ophthalmia. Minute foreign bodies in the eye were removed by the anterior route. Larger foreign bodies within the vitreous were removed through the sclera. In the case of exposure to mustard gas, if the eyes were washed immediately with sodium-bicarbonate solution very little damage resulted. Only the eyes that were bandaged were hopelessly lost. Such eyes should have been left uncovered.

Bullet wounds gave some strange results. Some soldiers were seen in whom the bullet went in one temple and out the other severing both optic nerves and causing little other damage. Bullet wounds that struck the occiput produced brain softening and bizarre visual fields. Wounds of one side of the head often produced symptoms referred to the other side. Many bullets went through the orbit without penetrating or cutting the eye, but such was the force of the projectile that the tissues of the globe were ruptured without penetration. It is unlikely that such a profusion of harrowing sights will ever be seen again since there is no longer such a thing as trench warfare.

Discussion. Dr. Meyer Wiener stated that he could not agree with the speaker about removing foreign bodies, no matter how small, by the anterior route. He said that he saw both Hirschberg and Haab use the giant magnet, and that much more damage was done than if the foreign bodies were pulled out through the sclera at the point nearest to the foreign body. He found that he could do better through the posterior route than by bringing the foreign body around to the front.

Dr. Mark Schoenberg asked if many cases of tetanus were seen in the last war.

Dr. Arthur Yudkin asked if there were any latent effects from the mustard gas.

Dr. Arthur Greenwood, in concluding, stated that many of the small foreign bodies were removed anteriorly because it saved time and did not require X-ray localization so often. He said that he had never seen a case of tetanus in the American army. He did see two cases in the British army. He said that 60 percent of cultures from wounds showed the Welch bacillus. He never saw any injury to the optic nerve from mustard gas. Hypopyon keratitis was almost never seen. Those corneal lesions due to a traumatic lagophthalmos were cared for by a conjunctival flap.

Personal experiences in eye surgery during the world war

Dr. Meyer Wiener of St. Louis said that he hoped that the Surgeon-General of the U. S. Army would profit by the mistakes made in the last war should another be inevitable. He was assistant to Dr. deSchweinitz in the Army School of Ophthalmology. Many who had registered as ophthalmologists had left a general practice and hoped to become oculists after a four-weeks' course. These and other political appointees were rapidly weeded out.

After the armistice he was stationed at

General Hospital No. 11, at Cape May, where much of the plastic repair work was done. With the assistance of Dr. Vilray Blair all patients received long and conscientious study before the actual repair was undertaken. Much of this work might have been avoided had there been time for better first-aid surgery at the front.

The Esser inlay and Esser outlay were of inestimable value in restoring sockets and in contractile entropion. One advantage of the buried graft is that it almost always takes in its entirety, and the results are obtained more quickly. Where there has been much destruction of tissue a full-thickness pedicle graft is much better. The work was not limited purely to the eye. Often the nasal and the frontal regions were included in the operative procedures. Catgut was not used for either sutures or ligatures. Silk was found to be cleaner and safer, became encapsulated, and caused no irritation.

Discussion. Dr. Percy Fridenberg said that in wartime there will be ophthalmologic problems involving both the military and civilian population. Gases, concussions, explosions, and blackouts all bring special problems in addition to direct injuries. A man who has lost an arm or a leg might still be useful, but a blind soldier is a total loss from the military standpoint. The special knowledge of the ophthalmologist is of tremendous importance. It stands to reason that in warfare where so much depends on the eyes and their equipment, many appliances and procedures will be invented to bring the important visual function to even greater degrees of efficiency in the service of our armed forces and to civilians also.

Repair of recent lid injuries

Dr. Henry Minsky read a paper on this subject and suggested the following procedure for repair of complete lacerations of the lid margin. A double-armed suture is passed in the plane of the gray line through both tips of the wound by placement of equal quadrant arcs. The lips of the marginal tears are then brought together into exact approximation by the first half of a surgeon's knot. The lacerated conjunctiva is repaired by buried catgut sutures. The mattress suture of the lids is completed by being brought through the gray line of the intact opposite lid to the skin surface and tied over a pig. A figure-eight suture is used when both lids are involved.

Discussion. Dr. Wiener reëmphasized the importance of the marginal suture. He said that he never found it necessary to go through a very elaborate procedure and that he got good approximation by taking a bigger bite than 2 mm. as suggested. He uses an ordinary half-curve needle with Corticelli spool silk. He uses A for ligatures, AA for corneal sutures, and B for tensile strength.

Dr. Henry Minsky in closing stressed the tying of the sutures snugly but not tightly to avoid sloughing from subsequent edema. He also suggested that overcorrection was important in the vertical wounds. Cutting out an elliptical portion of skin gives a tip at the margin instead of a notch and is preferable, since the ultimate result is better.

Sidney A. Fox,
Secretary.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

September 23, 1940

Dr. John L. Lordan, *president*

Physical requirements for naval aviation

Colonel Harrison, Army Flight Surgeon, was asked to speak. America now

has just over one hundred flight surgeons, he stated, and with the necessity for more pilots the number required to examine the vast number of applicants will exceed one thousand.

LIEUTENANT ALLMAN of the United States Navy spoke on the physical requirements for naval flying cadets. He said that many pilots trained under the Civil Aeronautics Administration were not acceptable in the Army or Navy due to higher standards of physical requirement.

REPAIR OF CORNEAL SCAR

DR. GEORGE KILGORE (by invitation) presented the pathology of corneal scars and their medical treatment. He then discussed the indications for corneal resection: (1) disfigurement of the eye by scar; (2) poor vision due to scar; and (3) painful corneal scars; that is, from recurrent ulcerations. A motion picture of the removal of a scar by the author was shown. He presented three case reports, good results having followed in each instance.

Discussion. Dr. George Landegger reported three cases in which the vision was improved following corneal resection. X-ray and radium therapy in the postoperative care was discussed.

INTRAOCULAR FOREIGN BODIES

DR. JOHN OSBURN presented some interesting case reports drawn from his many years of experience with intraocular foreign bodies. He compared removal of an intraocular foreign body by the anterior route, using the limbal section, with removal by the posterior route. The latter procedure was preferred in the cases in which there was no traumatic cataract.

Orwyn H. Ellis,
Recorder.

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CHANGES IN ASTIGMATISM

Injury or inflammation of the cornea may cause change in the shape of its surface, and so change the corneal astigmatism. Operative corneal incisions, for glaucoma or for cataract extraction, are expected to make marked changes in astigmatism. But aside from such instances of corneal traumatism, changes in corneal curvature are not expected. Astigmatism of the crystalline lens was first generally recognized by the difference it made between the corneal astigmatism and the total astigmatism of the eye. Apart from injury and disease, the cornea generally retains its shape from early adult age to the end of life.

The usual history of lens astigmatism is quite different. The lens continues to grow so long as it remains clear. Priestley Smith found it averaged one third more, in weight and in volume, at 65, than it did at 25 years of age. Such growth causes the prominence of the central part of the iris, and sometimes the shallow angle of the anterior chamber in advanced age, and has been recognized as a cause of glaucoma. But the growth of the lens has not been thought of as the common cause of change in the total astigmatism of the eye. In 1891 de Schweinitz, in his paper on "Astigmatism at different ages," pointed out that "astigmatism against the rule" was much more common in elderly patients. This has been observed by others, and may be

accepted as well established.

Corneal astigmatism in the lower animals has been observed by students of mammalian eyes. For them it may help in recognizing objects that stand up on the horizon. But with accurate macular vision, a human eye is more efficient without such astigmatism. Heredity has come to neutralize corneal astigmatism by increase of lens astigmatism. The wonderful optical accuracy of the eye has been reached by a process of evolution. The growth of the lens, in a shape to effect this, is the probable explanation of the change in lens astigmatism.

Changes in lens astigmatism have a very practical importance in the correction of errors of refraction. This idea has been emphasized recently by seeing in one day two cases in which lens changes brought back patients for whom correcting glasses had been previously prescribed, and had been worn, until lately, with complete relief.

Miss R. M., when in school at the age of 15 years, had been given: R. +3.25 D. sph. ⌯ −0.25 D. cyl. ax. 180°; L. +3.50 D. sph. ⌯ −0.50 D. cyl. ax. 170°. These were worn with complete relief. At the age of 21 years she was working in an office, making constant use of her eyes, but with a good light. She had developed a severe, constant headache. Her right eye showed no change in refraction, but her left eye now requires +4.25 D. sph. ⌯ −0.75 D. cyl. ax. 140°. This correction gave prompt relief from the headache.

Miss V. F., at 17, was relieved of headache by wearing R. and L. −0.50 D. cyl. ax. 180°. At 23 she was teaching and came for a return of headaches. These were relieved by wearing R. −0.75 D. sph. ⌯ −0.37 D. cyl. ax. 5°; L. + 0.37 D. cyl. ax. 87°. A year later she had a return of headache, and required for full vision and relief no change in the right eye, but in the left: −0.25 D. sph. ⌯ −0.50 D. cyl. ax. 3°.

In each of these cases the reëxamination and change of one lens gave relief. The changes in astigmatism had not made any increased demand for accommodation. It was simply on account of the difficulty of sustained coördination, when one eye required a diminished accommodation to avoid blurring of vision, that the new lens was required. Such changes in refraction may not be common, but they are easily overlooked. No textbook has pointed out the danger of disability from decreased need of accommodation in one eye. Most accounts of astigmatism imply that, once accurately corrected, it will not change; and when no change has occurred in corneal astigmatism, it is too often assumed that the total error of refraction remains unchanged.

Edward Jackson.

WHAT TYPE OF CATARACT OPERATION?

In one of our western states, a form issued by the public welfare department for use by ophthalmologists recommending an operation for the relief or prevention of blindness (in most cases a cataract operation) carries a question as to what type of operation is proposed.

Most surgeons making out these forms reply by specifying whether the operation contemplated is intracapsular or extracapsular extraction. One surgeon, having indicated on the preceding line of the form that the condition to be dealt with was cataract, gave the type of operation proposed as "cataract extraction." Being asked which type of extraction he expected to use, he answered that he usually made this decision at the time of operation, being guided by the condition encountered after preparation of the pa-

tient; or even, at the last, by the possibility or impossibility of removing the lens in its capsule.

There can be little question as to the brilliancy of the results obtained after uncomplicated extraction of the lens within its capsule. But some of the worst results in cataract surgery follow unsuccessful attempts to perform the intracapsular operation. The expert operator who more or less habitually undertakes the intracapsular procedure may be able so to control his technique that even after rupture of the capsule he can insure a fair or good result in most instances. But the prospect of failure in such an attempt argues against the use of the intracapsular method by those who for various reasons are less skillful, or less adequately prepared to deal with emergencies.

"There is," says Wright, "an objection to the argument that the occasional operator will find the forceps method equivalent to a well-planned capsulotomy —even if he tears the capsule. To anyone who thinks, the fallacy is obvious. It is not equivalent, and the occasional operator may find himself in the nasty position of having both a torn zonule and a torn capsule if he sets out with the idea that they are equivalent."

It may be proper to inquire how general is the practice of calling a cataract operation "extracapsular" although it began as "intracapsular." To what extent are the statistics of cataract operations rendered misleading by such a practice? To give both types of operation their due, results good or bad should be attributed to the intracapsular method when this has been attempted.

The use of statistics in scientific investigation demands complete intellectual honesty. Nowhere is this necessity greater than in dealing with cataract surgery.

W. H. Crisp.

DORYL IN GLAUCOMA

The search for more adequate and satisfactory treatment of glaucoma has only in recent years led to the use of drugs other than pilocarpine and eserine. One of the most promising of the newer drugs is doryl.

Doryl (carbaminoylcholine) is a synthetic choline ester that acts principally by stimulation of the parasympathetic nerves. Thus, in the eye the blood vessels are dilated, the pupil is constricted, there is spasm of accommodation, and the intraocular tension is lowered.

In 1933, Velhagen reported that instillations of an aqueous solution of doryl lowered the intraocular tension in cases of glaucoma. Shortly thereafter Miloro, Fontana, Hynie, and De Sanctis found doryl preferable to pilocarpine in the treatment of glaucoma simplex, but both Wilenkin and Magitot stated that the two drugs had approximately the same effect. Molitor noted that the effect of doryl varied with the mode of administration. Such differences of opinion continued, and the drug did not come into general use in the treatment of glaucoma. Recently, however, Swan found that aqueous solutions of doryl are poorly absorbed through the cornea, and this may account for the variance of opinion regarding its action. Swan believes that if the drug is combined with a surface-tension-reducing agent such as zephiran, absorption is good and its action is dependable. Doryl in a concentration of 1.5 percent in a 1:3,500 zephiran solution has been found by Swan to be more efficacious in the reduction of elevated intraocular tension in cases of glaucoma simplex than is pilocarpine. As with other drugs the tension is not controlled in all patients, but usually doryl acts to reduce the intraocular pressure to a lower level than does pilocarpine or eserine. Doryl is used in the same manner and of course

has the disadvantage of any home treatment; namely, dependency on the patient to use it regularly and in the proper manner. Some patients experience pain and headache during the first two or three days of treatment, but this is usually negligible and soon disappears. The drug is expensive at present; however, the price may be reduced if the drug comes into general use. At present doryl seems to be the drug of preference in the majority of cases of glaucoma simplex.

C. S. O'Brien.

THE 1941 ACADEMY MEETING

The forty-sixth annual session of the American Academy of Ophthalmology, and Otolaryngology, held at the Palmer House in Chicago, October 19th to 23d, has taken its place in history. More than 2500 members and guests attended. Judging from opinions expressed on all sides the meeting was thoroughly enjoyed by all. As usual every minute of the time was accounted for, and there are many who regret that the number of hours in a day is cruelly limited. The sessions have been aptly compared to a three-ring circus. This comparison is justified by the spectacle of formal papers, instruction hours, scientific moving pictures and exhibits, fun and feasts, excitement and glamor, all crowded into four days and sometimes going on all at the same time. Such activities can be profitable only when well organized. The Academy's method of organization and management commands the admiration and respect of all of its members. That they find it profitable is attested by the ever-increasing roll of membership.

It is but natural that this year the assembly should take on a defense coloring and an international tinge. The guest of honor was Dr. Perry G. Goldsmith of Toronto, Canada, and the honor guest, Sir Harold Gillies of London, England. The president, Frank R. Spencer, pledged the Academy, its resources, and membership to the service of our country. The Teachers' Section held a Defense Program dinner at which Colonel Louis H. Bauer, M.C., U.S.A., of Hempstead, Long Island, discussed aviation medicine, Sir Harold Gillies showed lantern slides and movies of plastic surgery in this war, Dr. Irvin Abell, past president of the A.M.A. and Coördinator-Surgeons-General of the Armed Services, U.S.A., spoke on the role of the physician in the national defense program. Splendid movies of aviation training in the Navy and Marines concluded an interesting and stirring evening.

The scientific aspects of the meeting will be discussed at another time. Here there is but space to say that the Academy program was of better than average caliber, and the scientific exhibits far superior to those in the past. The scientific motion pictures attracted such large crowds that the hall could not accommodate all. The popularity of this feature will no doubt lead to better facilities in the future.

The evening of diversion, a forty-niner party, swarmed with good nature. One looked in vain for dignity and bedside manners. The banquet, a feature that seems to be growing in popularity too, was well attended by the members, who had recovered their majesty and poise, accompanied by their ladies and guests. The president of the University of Colorado, Robert L. Stearns, gave an address on the merits of democracy that quickened the spirit and refreshed the soul fatigued by propaganda. The culture of his oratory and thoughts was an inspiration to the audience. One's animal spirits were simultaneously refreshed by good Chicago beef and other Lucullan items.

Dr. James A. Babbitt, emeritus pro-

fessor of clinical otolaryngology at the University of Pennsylvania School of Medicine, and associate professor of otolaryngology in that university's graduate school of medicine, Philadelphia, was named president-elect of the Academy. Dr. Babbitt will take office January 1, 1943. The present president-elect is Dr. Ralph I. Lloyd, Brooklyn, who will assume office on January 1, 1942.

Vice-presidents elected are Drs. Walter Theobald, Chicago; Forrest J. Pinkerton, Honolulu, Hawaii, and Francis E. Le Jeune, New Orleans. Dr. Secord H. Large, Cleveland, was reëlected comptroller and Dr. William P. Wherry, Omaha, Nebraska, executive secretary-treasurer.

Derrick Vail.

BOOK NOTICE

VISION—A STUDY OF ITS BASIS. By S. Howard Bartley, Ph.D. Clothbound, 337 pages, 78 illustrations, with an historical perspective by Dr. Edwin G. Boring of Harvard University. New York, D. Van Nostrand Company, 1941. Price $3.50.

This treatment of vision is directed towards "a correlation between experience and neurophysiology," and towards an explanation of how the visual pathway accomplishes this correlation. Dr. Boring, in contrasting the present work with the psychophysical view of Troland and with the psychophysiological methods of Hecht and of Graham, comments that Bartley's approach "properly represents a new physiological phase." Within prescribed limitations, the development of the subject proceeds along the lines of current research in sensory physiology. In general, the functioning of the neural mechanisms associated with vision is discussed from general rather than detailed anatomical bases. The scope of the discussions is indicated by the following sequence of abbreviated chapter titles: brightness discrimination, stray light, sense-cells, blind spot, flicker, perception of movement, adaptation, neural interaction, contour, electroretinogram, optic-nerve discharge, and cortical response. The presentation of the subject matter is more in outline than in detail, with the exception of descriptive accounts of researches by the author and by some other workers in similar fields. In this respect, it would appear that the limitation of material discussed is somewhat severe.

The last chapter separates, with considerable clarity, what is known and what is unknown with respect to certain sensory phenomena. A brief summary dealing with sense-cell behavior, the bipolar and ganglion cells, optic-nerve discharge and the response of the optic cortex is followed by a short discussion of some of the major problems of vision in this specific field. This carefully written treatise should be of particular interest to those engaged in visual research.

Matthew Luckiesh.
Frank K. Moss.

ABSTRACT DEPARTMENT

EDITED BY DR. WILLIAM H. CRISP
ASSISTED BY DR. GEORGE A. FILMER

Abstracts are classified under the divisions listed below, which broadly correspond to those formerly used in the Ophthalmic Year Book. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. General methods of diagnosis
2. Therapeutics and operations
3. Physiologic optics, refraction, and color vision
4. Ocular movements
5. Conjunctiva
6. Cornea and sclera
7. Uveal tract, sympathetic disease, and aqueous humor
8. Glaucoma and ocular tension
9. Crystalline lens
10. Retina and vitreous
11. Optic nerve and toxic amblyopias
12. Visual tracts and centers
13. Eyeball and orbit
14. Eyelids and lacrimal apparatus
15. Tumors
16. Injuries
17. Systemic diseases and parasites
18. Hygiene, sociology, education, and history
19. Anatomy, embryology, and comparative ophthalmology

1

GENERAL METHODS OF DIAGNOSIS

Asarova, H. C. **Van Lint's test in metastatic ocular tuberculosis.** Viestnik Opht., 1941, v. 18, pt. 3, p. 281.

This test for capillary fragility was applied to a series of patients with ocular tuberculosis, to determine if the test had any value in differentiation between a hemorrhagic and an exudative process. The conclusion is that the test is equally positive in both types of pathologic process. Ray K. Daily.

Evans, J.N., and Singer, E. **Fluorescence microscopy applied to ocular tissues.** Arch. of Ophth., 1941, v. 25, June, pp. 1007-1019.

By utilizing filtered ultraviolet radiation, various substances that have a similar appearance in white light may be readily differentiated, owing to their characteristic fluorescence. A special fluorescent staining technique and the fluorescence microscope are described. The fluorescence of freshly frozen ocular tissues was examined in a dark field. A strong bluish autofluorescence was observed in Descemet's membrane, the lens, and the sclera. The other tissues showed fluorescence to a considerably lesser degree. Fluorescence could be imparted to the nonfluorescent tissues and the color of the fluorescent tissues could be changed by fluorochroming, which is a process similar to histologic staining. Fluorochroming consists in application of greatly diluted, highly fluorescent compounds (dyes as well as colorless substances) to the tissues. All substances used for fluorochroming in the present investigation were classified according to their electrical charge because, irrespective of their chemical characteristics, similar results were obtained with all members of the negatively charged group, and the positively charged substances always showed diffuse staining without any cell differentiation. The negatively charged substances imparted fluorescence to the corneal epithelium, to the ciliary body, and to the optic

nerve. Among these substances, rivanol stained the nuclei selectively and thioflavine S stained the entire tissue. Small droplets of vitamin A, which has a strong greenish-yellow fluorescence, could be identified in the retina. In the choroid of the cat a strong yellow fluorescent substance was seen. The fluorescence of this substance was uninfluenced by exposure to ultraviolet rays. The changes found are illustrated by photomicrographs, and a schematic drawing of the eye is presented showing the electrical charge of the various layers. J. Hewitt Judd.

Friedman, Benjamin. **A method of procuring a narrow beam of light for ophthalmoscopy.** Arch. of Ophth., 1941, v. 25, June, pp. 1032.

A very narrow slit may be obtained by gluing two half circles of thin copper foil over the top of the metal cap which is placed over the condensing lens of the ophthalmoscope, so that the straight edges project over the original diaphragm opening. J. Hewitt Judd.

Lasky, M. A. **Simulated blindness.** Arch. of Ophth., 1941, v. 25, June, pp. 1038-1049.

Simulated blindness of one eye can be demonstrated objectively and subjectively. The objective method consists of close observation of direct and consensual pupillary reactions and checking of the ocular movements in relation to the visual axis. The subjective methods of detecting simulated blindness are numerous and depend on the principles of optics and physiology. Numerous tests of both methods are described in which plano and concave mirrors, prisms, screens, visual-acuity charts, stereoscopes, diaphragm apparatus, and polaroid filters are used. J. Hewitt Judd.

Luckiesh, M., and Moss, F. **The Luckiesh-Moss test-chart illuminator.** Amer. Jour. Ophth., 1941, v. 24, Sept., pp. 1061-1062.

2

THERAPEUTICS AND OPERATIONS

Beyer, W. **The success and retention of polyviol implants.** Klin. M. f. Augenh., 1941, v. 106, March, pp. 272-279.

A new artificial material consisting of polyvinyl alcohol, water, and Congo red (which forms an elastic mass) has been used successfully for implants following enucleation. In a high percentage of the cases the implant was retained without complications. A few cases showed very violent inflammation of the conjunctiva and the surrounding tissues, but after the inflammation subsided the implants were retained. (12 illustrations.) Gertrude S. Hausmann.

Cecchetto, Ezio. **Tioseptal in ophthalmology.** Rassegna Ital. d'Ottal., 1941, v. 10, Jan.-Feb., pp. 133-141.

Tioseptal is para-amino-phenyl-sulphamido-pyradine. Its action is polyvalent and is especially efficacious against pneumococcus, meningococcus, and gonococcus. The author uses the drug for a week preceding ocular operations and finds it especially valuable where the conjunctiva is not healthy. It is also effective in ocular wounds, trachoma, abscesses, and dacryocystitis. Eugene M. Blake.

Chetverikov, V. I. **Streptocide in ocular infections.** Viestnik Opht., 1941, v. 18, pt. 2, p. 174.

A review of the literature and a report of the author's own clinical experience with the drugs of the sulfanilamide group used internally and locally. The conclusions are that solutions and

ointments are indicated locally in diseases of the conjunctiva and the lids. In corneal ulcers the powder should be used locally and the drug taken internally. The drug was found most effective in phlegmons of the lacrimal sac. Ray K. Daily.

Fontana, L. **Bee venom in ophthalmology.** Rassegna Ital. d'Ottal., 1941, v. 10, Jan.-Feb., pp. 116-131.

The use of bee venom in general diseases, especially rheumatic, is discussed, and the chemistry, so far as is known, is reviewed. The author used the venom in two different forms, one called "Trocoid," and another called "Forapin," the latter containing a snake venom in addition to the bee venom. Sixty-eight patients with various pathologic eye conditions were treated. Marked relief from pain was generally noted, and more rapid healing of corneal ulcers was observed.

Eugene M. Blake.

Foss, Bjorn. **Is a K-avitaminosis present in cases of postoperative intraocular hemorrhage?** Acta Ophth., 1941, v. 19, pt. 1, p. 15.

Hematologic studies answer this question in the negative, except for sprue cataract in which prothrombin in the blood is reduced; in such cases injections of vitamin K should precede surgery. Ray K. Daily.

Goldsmith, J. **A combination curet and suction spoon for evisceration of the eyeball.** Arch. of Ophth., 1941, v. 25, June, pp. 1032-1033.

To expedite the removal of the contents of the eye, the evisceration curet is attached to a brass tube which acts as its handle and to which rubber tubing connected with the suction apparatus may be attached.

J. Hewitt Judd.

Jendralski, Felix. **Results of forming a plastic socket after removal of the eyeball.** Klin. M. f. Augenh., 1941, v. 106, March, pp. 279-283.

In 13 patients aged 1 to 46 years implants of polyviol were used following enucleation. In 12 of these cases the implant was retained without complications. (2 illustrations.)

Gertrude S. Hausmann.

Loewenstein, Arnold. **Argyrosis of conjunctiva, cornea, and tear sac.** Brit. Jour. Ophth., 1941, v. 25, Aug., pp. 360-369. (See Section 16, Injuries.)

Lorenz, Roland. **The use of sulphonamides in diseases of the eye.** Klin. M. f. Augenh., 1941, v. 106, March, pp. 302-306.

In cases of acute and recurrent iritis, especially of gonorrheal origin, no certain results could be obtained from treatment with the sulphonamides. In gonorrheal ophthalmia the use of the sulphonamides was successful. In cases of trachoma a prolonged treatment of 20 to 30 days with albucid gave very good results.

Gertrude S. Hausmann.

Scheie, H. G., and Souders, B. F. **Penetration of sulfanilamide and its derivatives into aqueous humor of eye.** Arch. of Ophth., 1941, v. 25, June, pp. 1025-1031.

The absorption of sulfanilamide and three derivatives, and their penetration into the aqueous humor of the eye were studied in cats. Sulfanilamide and sulfapyridine readily penetrated into the aqueous humor. Sulfathiazole attained very small concentrations in the aqueous humor, as compared with its concentrations in the blood. Therefore, the use of sulfathiazole in the treatment of intraocular infections seems contraindicated. The ineffectiveness of

sulfathiazole against intraocular infection was observed in three cases: one of ring abscess of the cornea, another of penetrating wound of the cornea, and a third of postoperative cataract infection. A new sulfanilamide derivative, sulfadiazine, was found to diffuse into the aqueous humor in concentrations comparable to those observed with sulfanilamide and sulfapyridine.

J. Hewitt Judd.

Youfa, H. Z. **Experimental investigations of consensual ocular desensitization to tuberculin.** Viestnik Opht., 1941, v. 18, pt. 1, p. 25.

The objective of this laboratory investigation was to determine the reaction of the untreated eye to desensitizing treatment of the fellow eye. The report shows that both eyes are affected by desensitizing treatment of one eye. This bilateral reaction, Youfa believes, is not a part of a general process in which the entire organism participates, but a selective desensitization of paired organs. On the basis of these experimental results, he envisages the clinical instillation of tuberculin into the healthy eye with the object of influencing the diseased eye, and at the same time avoiding a provocation of the inflammatory process.

Ray K. Daily.

Zamkovskii, I. G. **Oxygen therapy of the eyes.** Viestnik Opht., 1941, v. 18, pt. 1, p. 71.

The author reports good results with subconjunctival and retrobulbar injections of oxygen, in various diseases of the eye.

Ray K. Daily.

3

PHYSIOLOGIC OPTICS, REFRACTION, AND COLOR VISION

Crozier, W. J., and Wolf, E. **Theory and measurement of visual mechanisms. 4. Critical intensities for visual flicker, monocular and binocular.** Jour. Gen. Physiology, 1941, v. 24, March 20, p. 505.

A quite complete and technical report of the comparison of monocular and binocular critical flash intensities for recognition of flicker.

Robert O. Sherwood.

Eggers, Harry. **Two new visual-test charts.** Arch. of Ophth., 1941, v. 26, July, pp. 71-78.

The author discusses the theoretic basis for visual-test charts and astigmatic fans and dials. He describes two charts in which all letters, ordinarily curved or slightly curved, are drawn with straight lines. To these are added broken circles with double breaks and two astigmatic fan dials. A chart of smaller astigmatic dials consists of six variously placed figures, each with two pairs of lines at right angles to each other. The lines subtend a one-minute angle at 40 feet. The patient selects the pair of lines that is darkest and most clearly defined and then decides when cylinders inserted into the trial frame equalize the two pairs of lines in blackness and in definition.

J. Hewitt Judd.

Fieandt, Olof. **A case of transitory myopia, following radium irradiation.** Acta Ophth., 1941, v. 19, pt. 1, p. 11. (See Section 16, Injuries.)

Harman, N. B. **Testing night vision.** Brit. Med. Jour., 1941, April 26, p. 636.

The author discusses the method of testing night vision devised by Wright and based on the detection of test letters illuminated by a radioactive substance. Harman disagrees with the method, largely because of the difficulties in standardization. He describes a

test based on the ability of the subject to correctly count white discs half an inch in diameter on a black background. The test card is illuminated by a standard candle placed in a box five meters from the test object. In this way a standard illumination of 1/269 foot-candle is obtained. The subject is first tested at five meters and then is allowed to approach the test objects until he can accurately count them.

John C. Long.

Jancke, G. **The origin of myopia.** Klin. M. f. Augenh., 1941, v. 106, March, pp. 264-272.

This report of new examinations of identical twins confirms the heredity theory of myopia. The author's comparison of a series of identical twins with other twins in similar occupations indicates the influence of heredity and proves that close work does not increase the myopia. (2 tables.)

Gertrude S. Hausmann.

Mihalyhegyi, G. **On the curvatures of the eyeball and making casts of the living globe.** Ophthalmologica, 1941, v. 101, May, p. 290. (See Section 6, Cornea and sclera.)

Preston, F. E. **Distance object test for children.** Brit. Jour. Ophth., 1941, v. 25, Aug., pp. 357-360.

Few children's charts conform with accuracy to the Snellen principle. The author has therefore experimented in order to achieve a higher degree of accuracy. The resultant chart consists of 32 objects, those being discarded which are not easily interpreted by the greatest number of children. (Figures.)

D. F. Harbridge.

Rauh, Walter. **The color field in experimental night blindness.** Graefe's Arch., 1940, v. 141, pts. 4 and 5, pp. 545-548.

A narrowing of the color field for yellow was found in ten men who had been on a vitamin-A-free diet for about six months. The degree of narrowing paralleled the degree of night blindness, and when most severe lay within or crossed the border of the field for green.

Frances C. Cogan.

4

OCULAR MOVEMENTS

Cordes, F. C. **The San Francisco orthoptic laboratory.** Amer. Jour. Ophth., 1941, v. 24, Sept., pp. 1057-1060.

Cushman, B., and Burri, C. **Convergence insufficiency.** Amer. Jour. Ophth., 1941, v. 24, Sept., pp. 1044-1052.

Feldman, J. B. **Clinical observations on the treatment of squint.** Arch. of Ophth., 1941, v. 26, July, pp. 38-56.

This report is based on the study of 56 private and 72 dispensary patients. Each group received orthoptic treatment by a trained technician under the author's supervision. There were 50 patients with alternating squint (4 with alternating divergent squint) and 6 with unilateral squint. The dispensary group consisted of 36 patients with alternating squint (3 with alternating divergent squint) and 34 with unilateral squint. The results are recorded under two separate headings, and the variations in the findings as to the anomalies in these two groups possibly explain how it happens that two observers arrive at somewhat different conclusions for the same entity. It was found that two or three lessons a week at the clinic gave more favorable results than single weekly lessons given

to private patients. The predisposing factors and the phenomena usually thought to accompany squint are discussed, requirements preliminary to orthoptic treatment are outlined, and the superiority of atropine to homatropine for mydriasis is shown. The obstacles to orthoptic training are noted with their incidence in the groups examined and the percentage of patients finally cured of each particular anomaly. Perimetric studies of amblyopia, alone or combined with squint, are given for a few cases in which uniocular fixation was used. A chart used in addition to the detailed history is shown by which the ophthalmologist can quickly note the cosmetic effect of the treatment. Based on the report of the findings in six children with stubborn squint examined by the neuropsychologic department, the author believes that in a case of squint, particularly of the alternating type, it is advisable to make a complete psychologic examination before attempting orthoptic treatment. This might obviate spending two or three years treating a condition in which one could obtain the same cosmetic result in much less time by surgical intervention. Orthoptic treatment is of value in that it often overcomes an obstacle not remedied by surgical means. It supplements normal physiologic function with cosmetic cure when possible.

J. Hewitt Judd.

Filippi-Gabardi, Eugenio. **Disturbances of the extrinsic ocular motility in patients affected by hyperthyroidism.** Riv. Oto-Neuro-Oft., 1940, v. 17, Nov.-Dec., pp. 564-573.

A woman of 31 years with severe general asthenic tremor of the limbs and slight enlargement of the thyroid showed a slight internal deviation of both eyes apparently due to an abnormal contraction of the internal recti muscles. Convergence was accomplished normally, but the eyes had a tendency to go back to the position of rest. Diplopia test with the red glass showed an irregular homonymous diplopia in every direction of the gaze, which could not be corrected by prisms. The writer considers such an oculomotor dysfunction to be an expression of tonogenic disturbance induced not by a paralytic phenomenon but by thyroid toxicosis or disturbance of the vegetative tonic innervation. (Bibliography.) M. Lombardo.

Grimm, R. **Discussion of the treatment of strabismus.** Klin. M. f. Augenh., 1941, v. 106, March, pp. 306-314.

Grimm discusses the different theories as to the origin of strabismus. Hyperphorias of more than 2° and esophorias of more than 6° cause faulty position of the eyeball. In rare cases operated upon before the tenth year it is possible not only to straighten the eyes but to get perfect fusion. However, if the fusion is not good the operative result is much worse than in cases operated upon at more advanced ages. Gertrude S. Hausmann.

Herzau, Werner. **Remarks on Meesmann's contributions to the operative treatment of vertical strabismus.** Klin. M. f. Augenh., 1941, v. 106, Feb., p. 225.

Reference is made to a publication in which Meesmann recommended myectomy of the inferior oblique muscle for such cases. (Amer. Jour. Ophth., 1941, v. 24, p. 463.)

Gertrude S. Hausmann.

Hitz, J. B. **The management of strabismus at the Milwaukee Children's**

Hospital. Amer. Jour. Ophth., 1941, v. 24, Sept., pp. 1019-1022.

Knauber, Edna. **What can be expected of orthoptic training?** Amer. Jour. Ophth., 1941, v. 24, Sept., pp. 1022-1024.

Lordan, J. P. **Orthoptic training—indications for and results.** Trans. Pacific Coast Oto-Ophth. Soc., 1940, v. 25, pp. 60-74.

The author comments on the formation of the American Orthoptic Council in 1938. The value of orthoptic training is stressed and the question of which patients are suitable for such training is discussed. Most patients receiving orthoptic training are strabismic. Occlusion of the master eye is practised until vision no longer improves in the poorer eye or until 20/50 is obtained. After all possible improvement in vision is attained, unstraightened eyes are operated upon and the operation is followed by more orthoptic training. The author's excellent results with surgery combined with orthoptics are noted. Orthoptics are considered more necessary in monocular convergent squint than in monocular divergent or alternating convergent squint.

Lawrence G. Dunlap.

Maxwell, J. T. **Muscle-balance determinations at the reading distance.** Arch. of Ophth., 1941, v. 26, July, pp. 98-101.

As a basis for constructive criticism of some methods employed in determining ocular efficiency at the reading distance, the author reviews physiologic laws involved in ocular convergence. He discusses the relation of accommodation to convergence, exophoria in near vision, accommodative and proximal convergence, and the near point of convergence.

J. Hewitt Judd.

Paneva, V. A., and Medvedev, H. I. **The dynamics of occupational nystagmus.** Viestnik Opht., 1941, v. 18. pt. 2, p. 189.

As a result of this study, made at the Institute of Hygiene and Work, the authors question the validity of the accepted view that the development of miners' nystagmus calls for a transfer of the worker to surface labor. The material studied shows that miners with compensated nystagmus may remain at their jobs and those with mild forms without disagreeable subjective symptoms may remain in the mines doing lighter work, without danger to their vision. Ray K. Daily.

Sverdlick, José. **Etiopathogenesis, orthoptic training, and re-education of functional strabismus.** Arch. de Oft. de Buenos Aires, 1939, v. 14, Nov., p. 955.

The muscular, neurologic, Donders, and Worth theories are discussed in detail. The importance of the problem of strabismus in children is stressed. The author describes the method of treatment carried out in the ophthalmologic clinic at the Rawson Hospital.

Pre-orthoptic treatment comprises the early correction of refractive error and the treatment of amblyopia and suppression, thus paving the way for the visual re-education of the patient. The most effective treatment of amblyopia is occlusion of the fixing eye. General treatment must not be neglected; and removal of all foci of infection and administration of calcium and vitamins must be undertaken whenever indicated. Deviations greater than 25 degrees call for operative measures. Orthoptic training is used preopera-

tively or postoperatively according to the age of the patient and the status of the binocular vision. The necessity for close coöperation between ophthalmologist, patient, and family is emphasized. Plinio Montalván.

5

CONJUNCTIVA

Barsoum, Fahim. **Bilharzial granuloma of bulbar conjunctiva.** Giza Mem. Ophth. Lab., 1938, 13th rept., p. 50.

A mass of granulation tissue under the bulbar conjunctiva showed the presence of bilharzial ova.

Edna M. Reynolds.

Blumenfeld, H. A. **Tuberculosis of the conjunctiva.** Viestnik Opht., 1941, v. 18, pt. 2, p. 203.

A review of the literature with a report of three cases, two of primary tuberculosis, and one of metastatic involvement in pulmonary tuberculosis.

Ray K. Daily.

Dering, S. A. **The effect of tuberculin allergy on the development and course of the trachomatous process.** Viestnik Opht., 1941, v. 18, pt. 2, p. 167.

A review of the literature, and a report on the Pirquet reaction in 54 trachoma patients. The records show a positive reaction of various degrees in 69.26 percent. Most of the cases of severe pannus were found in the tuberculin-positive group. Treatment with tuberculin reacted favorably on the pannus. The author concludes that allergy is a factor in the appearance of severe pannus, that allergy is caused by tuberculous infection, and that there is an antagonism between pulmonary tuberculosis and severe trachoma.

Ray K. Daily.

Elles, N. B. **Rhinosporidium seeberi infection in the eye.** Arch. of Ophth., 1941, v. 25, June, pp. 969-991. (See Amer. Jour. Ophth., 1941, v. 24, June, p. 720.)

Kamel, Sabri. **Leproma of limbus.** Giza Mem. Ophth. Lab., 1938, 13th rept., p. 57.

Two avascular yellowish masses, one at the lower right limbus of each eye, gradually increased in size over a period of three years. There was no conjunctival congestion and no ulceration or opacity of the cornea.

Edna M. Reynolds.

Krasnov, M. L. **Two clinical case reports: arachnodactylia and bilateral dislocation of the lens; and conjunctival cyst after enucleation.** Viestnik Opht., 1941, v. 18, pt. 2, p. 198. (See Section 9, Crystalline lens.)

Lugossy, Gyula. **Up-to-date treatment of gonorrheal ophthalmia.** Klin. M. f. Augenh., 1941, v. 106, March, pp. 295-301.

Ultraseptyl, a sulphonamide preparation, produced no toxic effects. It may be combined advantageously with foreign-protein therapy.

Gertrude S. Hausmann.

Lukić, Djordje, J. **The treatment of gonorrheal conjunctivitis with septoplix given orally.** Klin. M. f. Augenh., 1941, v. 106, Feb., pp. 198-208.

Septoplix, a para-aminophenylsulfamide, was given in 22 cases of gonorrheal conjunctivitis. The gonococci in most of the cases disappeared within two days, but the treatment was continued for several more days to avoid exacerbations, which had occurred in two cases where the drug was discontinued prematurely. No toxic symp-

toms (anemia, rashes, or hemoglobinuria) were observed. (2 tables.)

Gertrude S. Hausmann.

McKelvie, A. R., Kirk, R., and Holder, H. J. **Observations on the chemotherapy of trachoma.** Amer. Jour. Ophth., 1941, v. 24, Sept., pp. 1035-1043.

Metivier, V. M. **Bitôt's spots in Trinidad.** Amer. Jour. Ophth., 1941, v. 24, Sept., pp. 1029-1034.

Neher, E. M. **A new method for transplanting pterygium.** Trans. Pacific Coast Oto-Ophth. Soc., 1940, 28th mtg., p. 152. (See Amer. Jour. Ophth., 1939, v. 22, Aug., p. 938.)

Ochapovskaja, H. V. **Parinaud's syndrome and tuberculosis of conjunctiva.** Viestnik Opht., 1941, v. 18, pt. 3, p. 278.

A review of the literature and a report of two cases. Parinaud's syndrome is the expression of the anatomophysiologic character of the conjunctiva, and its reaction to an infection which tends to invade the blood and meet the lymphoglandular barrier. It represents a macroscopic syndrome, of varied microscopic appearance, and of varied etiology. Ray K. Daily.

Ochi, S. **Etiologic investigation of trachoma.** Ophthalmologica, 1941, v. 101, May, p. 272; and June, p. 355.

For a long time the author has used serial passage of virus through rabbits' testes for his studies on the etiology of trachoma. Specific cell inclusion always appears when the testes react properly to inoculation with virus. These cell inclusions appear in two distinct forms, numbered 1 and 2 by the author, but transitional forms are also observed. Form 1 develops from typical elementary bodies, while form 2 arises from a spore-like unit described by the author.

F. Herbert Haessler.

Reis, Carolina. **Serologic reactions in trachoma.** Viestnik Opht., 1941, v. 18, pt. 3, p. 292. (See Amer. Jour. Ophth., 1940, v. 23, Oct., p. 1188.)

Suárez Gabard, Santiago. **The action of sulfanilamide derivative 693 (Dagenan) in gonococcic conjunctivitis.** Arch. de Oft. de Buenos Aires, 1939, v. 14, Dec., p. 1037.

Five cases of gonococcic conjunctivitis are reported in which sulfanilamide derivative 693 (Dagenan) was used. An average dose of 6 to 7 centigrams per kilogram of body weight was used. No intolerance to the drug was observed. In order to evaluate the therapeutic efficacy of this substance, no local treatment was used. The author arrives at the following conclusions: The bactericidal action of Dagenan is so effective that it can be used alone in the treatment of gonococcic conjunctivitis in spite of its severity. Twenty-four to 48 hours after administration of the drug the clinical picture has almost subsided, and the gonococci disappear from the conjunctival discharge between the second and sixth days. If the treatment is discontinued before the conjunctiva returns to normal there may be recurrences in spite of negative bacteriology.

Plinio Montalván.

Tatar, Josef. **Experiences with a non-irritating colloidal protein silver preparation in ophthalmology.** Klin. M. f. Augenh., 1941, v. 106, Feb., pp. 220-224.

Targesin, a colloidal protein silver

preparation, has apparently proven effective in treating pneumococcic infections. Acute conjunctivitis and trachoma reacted favorably. The preparation can be used in drops (3 to 25-percent solution), and in the form of an ointment.

Gertrude S. Hausmann.

Wilson, R. P. **Cholesterin degeneration of cornea and palpebral conjunctiva.** Giza Mem. Ophth. Lab., 1938, 13th rept., p. 83. (See Section 6, Cornea and sclera.)

Wilson, R. P. **Koch-Weeks and gonococcal ophthalmias.** Giza Mem. Ophth. Lab., 1938, 13th rept., p. 103.

The course of Koch-Weeks conjunctivitis did not appear to be shortened to any definite extent by the administration of sulphonamides. Cases of acute gonococcal conjunctivitis responded most satisfactorily to treatment with Dagenan. In most cases there was no edema or discharge after 24 hours treatment.

Edna M. Reynolds.

Wilson, R. P. **Lipoid degeneration of cornea and palpebral conjunctiva.** Giza Mem. Ophth. Lab., 1928, 13th rept., p. 81. (See Section 6, Cornea and sclera.)

Wilson, R. P. **Retention cyst of serous glands in caruncle.** Giza Mem. Ophth. Lab., 1938, 13th rept., p. 47.

A case report.

Wilson, R. P. **Treatment of trachoma and the acute ophthalmias with sulfonamide compounds.** Giza Mem. Ophth. Lab., 1938, 13th rept., p. 98.

Sulfonamide treatment of palpebral trachomatous lesions in fifty children, none of whom showed active pannus or keratitis, produced only slight general improvement. In 18 cases of young adults suffering from recurrent attacks of trachomatous keratitis, excellent results were obtained. After two weeks treatment corneal infiltrations were resolved and subjective symptoms disappeared. In the healing of the lids it was necessary to continue the treatment together with local applications for a considerable time after the corneal lesions had cleared up.

Edna M. Reynolds.

6

CORNEA AND SCLERA

Bürki, E. **Cystine disease in early childhood, with particular reference to eye findings.** Ophthalmologica, 1941, v. 101, May, p. 257; and June, p. 331.

By 1937 only seven cases of cystine disease had been reported, and in 1940 two more were added. Among 250 cases of renal rickets or renal dwarfism, some cystine diseases may have occurred. The author reports in utmost detail the results of his exhaustive studies of a young child with renal dwarfism and cystine disease, brought to him because the corneas were nebulous. The outstanding clinical manifestation was a progressive renal insufficiency in a seven-year-old boy who was 89.5 cm. long instead of the average 116 cm.

The eyes were free from irritation. The conjunctivas were strikingly pale and in the exposed portion dotted with a fine glistening yellowish punctate conglomeration of crystals. A slight nebula involving the entire cornea consisted of similar opacities many of which were extremely fine and a few somewhat larger. Peripherally they occupied the entire thickness of the cornea, while in the central portion the posterior third was relatively free. A piece of conjunctiva removed for biopsy gave reactions not incompatible with

cystine but proof was not unequivocal.

Autopsy findings confirmed the probable diagnosis, namely: renal dwarfism with cystine diathesis, severe rickets, and osteofibrosis. Particularly the epiphyses of the humerus, femur, ribs, and vertebrae were involved. There was chronic glomerular and interstitial nephritis and terminal uremia. The spleen was hyperplastic, with maximal cystine storage. There was great cystine storage in liver, lymph nodes, thymus, and gastro-enteric mucosa, and less in the testes.

In the eyes there was great cystine storage in the conjunctiva, cornea, episclera, and uvea, but only a little in the sclera. There was irregularity of the corneal lamellae and lymphoid infiltration about the crystal conglomerates in the uvea. In the retina and optic nerve were found edema, hemorrhage, and areas of degeneration, which were ascribed to uremia.

The pathogenesis is discussed at length. It is concluded that the process is inadequately understood, but probably results from absent or abnormal catabolism or diaminization of cystine. The immature organism is likely unable to cope with this flood of abnormal metabolites, and the kidney gives way. It is pointed out that the eye findings are essential to a clinical diagnosis.

F. Herbert Haessler.

Čavka, Vladimir. **Contribution to familial degeneration of the cornea.** Klin. M. f. Augenh., 1941, v. 106, March, pp. 348-352.

A case of degeneration of the cornea is reported, and the different forms of degeneration are discussed.

(2 illustrations.)

Gertrude S. Hausmann.

Cogan, D. G. **Bullous keratitis. With particular reference to the pathology of experimental corneal vesiculation.** Arch. of Ophth., 1941, v. 25, June, pp. 941-968.

The results of experimentation on cats' eyes support the theory that corneal vesicles and bullae develop because fluid is actively attracted into the epithelium through its anterior surface owing to an osmotic tension that is greater in the epithelium than in the tears. The histologic changes associated with experimental bullous keratitis and those observed in the clinical condition are basically similar. Swelling of the epithelium occurs with frequent vacuolation of the nuclei. The epithelium is loosened and in places separated by vesicles and bullae containing amorphous debris, probably composed of the contents of ruptured epithelial cells. This debris is gradually replaced by layers of spindle cells resembling those of connective tissue. Bullous keratitis represents a late stage of epithelial edema, and the mechanism by which tear fluid is attracted into the epithelium in bullous keratitis probably applies in other types of corneal edema as well. Special mention is made of the corneal changes occurring with cocainization and with recurrent corneal erosion. Edema of the corneal epithelium differs from the usual edema elsewhere in the body in that it is presumably dependent more on direct absorption of the tears than upon circulation of the blood. (Photomicrographs.) J. Hewitt Judd.

Fine, Max. **The present status of the corneal-transplant operation.** Trans. Pacific Coast Oto-Ophth. Soc., 1940, v. 25, pp. 75-95.

The author discusses the indications, techniques, and results of corneal transplantation. An interesting table accompanying the paper estimates that

of 238,000 blind persons in Russia, 117,000 (45 percent) are blind from leukoma and of 117,000 blind persons in the United States, 22,000 (19 percent) are blind from the same cause. It is further estimated that about one half of these are suitable for optical keratoplasty. The other half are unsuitable because of advanced age, secondary glaucoma, or retinal, uveal or optic-nerve diseases complicating the corneal disease. Corneal transplantation should never be done in an individual with one sound eye or in a one-eyed person whose vision is 1/10 or better, since the resultant postoperative visual acuity in the majority of successful cases is only about 1/10. In the successful cases the cornea must not be completely opaque. The importance of small doses of X ray to prevent postoperative vascularization and opacification of the transplant is stressed. Opaque grafts are due to having been inserted into opaque leucomas, incarceration of the iris, infections, corneal fistulas, or postoperative glaucoma.

Lawrence G. Dunlap.

Friede, R. **Central corneal fistula cured by keratoplasty after Majewski.** Klin. M. f. Augenh., 1941, v. 106, March, pp. 333-337.

A fistula at the center of the cornea following corneal ulcer was closed by keratoplasty, and the ulcer and surrounding tissue were excised with a trephine. The transplant, which was taken from an eye enucleated for chronic uveitis, did not remain clear, and this was attributed to the fact that the flap was from an inflamed eye.

Gertrude S. Hausmann.

Garkavi, P. A. **The anatomo-pathologic basis of permanent corneal opacities.** Viestnik Opht., 1941, v. 18, pt. 3, p. 284.

A correlation between the biomicroscopic and histologic pictures. They show that even in pronounced corneal opacities the entire corneal stroma is not replaced by scar tissue. Extensive cicatrization is seen only in staphylomas which are complicated by invasion of iris tissue. The opacity of leucomas is caused not by the cicatricial tissue alone, but also by the changed character of the remaining or regenerated corneal stroma. In most cases of parenchymatous keratitis, even with poor vision, there is little connective tissue and little stroma destruction. (Illustrations.) Ray K. Daily.

Grawitz, P. B. **At what temperature is the last trace of life in our tissues extinguished?** Graefe's Arch., 1940, v. 141, pts. 4 and 5, pp. 513-516.

Pieces of rabbit cornea were heated for one minute in dry air at temperatures of 180, 185, and 190°C. and then implanted in a host according to a technique previously described. In corneas heated to 180 and 185° the author found some residual cell structure which he interpreted as being a manifestation of life. Corneas heated to 190° were found to show no reaction.

Frances C. Cogan.

Gurvish, B. A. **Late results of the treatment of trachomatous pannus with silver nitrate.** Viestnik Opht., 1941, v. 18, pt. 1, p. 39.

Eleven brief clinical histories demonstrate the effectiveness of the author's therapeutic procedure. It consists of very thorough application of 2-percent silver nitrate to the conjunctiva and cornea. Ray K. Daily.

Kuvatov, G. G. **The role of the trigeminus in pathogenesis of keratitis.** Viestnik Opht., 1941, v. 18, pt. 1, p. 44.

A laboratory study on dogs leads to the conclusion that keratitis associated with injuries to the oculomotor is the result of involvement of the trigeminus. The biologic process probably consists in an interference with production of acetylcholine in the corneal tissue. This view is supported by the fact that the aqueous of dogs which developed experimental keratitis failed to give a reaction for acetylcholine. Early symptoms of beginning keratitis are contraction of the pupil and diminished corneal sensitivity. Ray K. Daily.

Mihályhegyi, G. **On the curvatures of the eyeball and making casts of the living globe.** Ophthalmologica, 1941, v. 101, May, p. 290.

In the course of contact-glass work, the author observed that scars of the cornea deformed not only that structure but also the sclera. That there is a connection between the curvatures of these tissues is also supported by an observation of Sanctis, who noted modified corneal curvature after diathermal coagulation of the sclera for retinal detachment. The author describes a slight modification of Csapody's tubes for making casts of the anterior segment.

F. Herbert Haessler.

Poštić, Svetozor. **Keratitis neuroparalytica following electrocoagulation of the gasserian ganglion.** Graefe's Arch., 1940, v. 141, pts. 4 and 5, pp. 559-560.

The author reports a case of keratitis neuroparalytica which appeared 36 hours after electrocoagulation of the gasserian ganglion for trigeminal neuralgia. Two months later the cornea was opaque, ulcerated, and vascularized. It was treated with some success by ultraviolet light, and Vogan's salve in concentrations of 10 to 20 percent.

Frances C. Cogan.

Reiser, K. A. **Histologic studies of herpetic inoculation keratitis in rabbits. Part 2. The cause of sensory disturbance.** Graefe's Arch., 1940, v. 141, pts. 4 and 5, pp. 339-357.

Histologic sections of rabbit corneas, some inoculated with "vaccine" and some with herpes virus, and sections of ulcus serpens in man show a leukocytic infiltration along the terminal nerve filaments. This suggests that the corneal nerves are anatomically susceptible to toxic and mechanical damage in the early stages of these diseases.

Herpetic keratitis differs histologically from vaccine keratitis and ulcus serpens in that there is a diffuse infiltration instead of a typical leukocytic ring in the cornea. In herpes the infiltration is densest at the limbus especially in the perineural spaces. There are small subepithelial necroses at the limbus and swollen lymph vessels near the corneal nerves. The author thinks that in herpes keratitis the sensory path from the cornea to the brain is blocked at the limbus, which would explain why the sensory disturbance is greater in proportion to the size of the lesion than in vaccine keratitis or ulcus serpens.

Frances C. Cogan.

Rones, Benjamin. **Pathologic conditions of the cornea. 2. The substantia propria.** Arch. of Ophth., 1941, v. 26, July, pp. 108-126.

The author reviews the anatomy of the substantia propria and discusses the general pathologic conduct, pigmentation, degeneration, wounds and repair, inflammation (both purulent and non-

purulent) and dystrophies. The histologic changes in the various conditions are shown by photomicrographs.
J. Hewitt Judd.

Sysi, R. **Molluscum contagiosum of the cornea.** Acta Ophth., 1941, v. 18, pt. 1, p. 25.

This is the first report of such a case. A small nodule, with central depression, was situated in the center of the left cornea of a three-year-old boy. He had lesions on the face and lid border. The lid-border growth showed histologically the typical molluscum contagiosum picture. The corneal lesion, because of its small size, could not be diagnosed with certainty, but the few cells that were shown in the preparation were of the molluscum-contagiosum type. Ray K. Daily.

Thomas, Maxwell. **Recurrent erosions of the cornea.** Southern Med. Jour., 1941, v. 34, April, p. 426.

The author has reviewed the literature in very considerable detail in the discussion of the etiology, pathology, symptomatology, prognosis, and treatment of this condition. He points out that recurrent corneal erosion is not a clinical entity but a syndrome. Treatment is directed toward removing all diseased epithelium and preparing the surface for the firm restoration of a healthy epithelial covering. Light curettage and application of strong iodine preparations are of considerable value, especially if herpes is a factor. Four illustrative cases are described.
John C. Long.

Vrolijk, M. **Corneoscleral cyst.** Acta Ophth., 1941, v. 19, pt. 1, p. 44.

A detailed report of a congenital epithelial cyst at the limbus of the left eye of a ten-year-old child; with a review of the literature relative to the etiology. (Illustration.) Ray K. Daily.

Walker, J. P. S. **A case of blue sclerotics.** Brit. Jour. Ophth., 1941, v. 25, Aug., p. 383.

Blue sclerotics occurred in both eyes of a 46-year old woman. There was also a history of long-standing deafness in the left ear, and a recent fracture of the femur. None of these conditions could be traced in other members of the family. The sight of the left eye was nearly gone. D. F. Harbridge.

Wilson, R. P. **Cholesterin degeneration of cornea and palpebral conjunctiva.** Giza Mem. Ophth. Lab., 1938, 13th rept., p. 83.

A segment of yellowish opacity extending at a deep level in the cornea from the limbus to the pupil showed the presence of a large branching vessel across the pupil. Along its branches lay numerous masses of cholesterin which covered the vessels and elevated the corneal epithelium. (Illustration in color.) Edna M. Reynolds.

Wilson, R. P. **Episcleral abscess.** Giza Mem. Ophth. Lab., 1938, 13th rept., p. 84.

An inflammatory nodule above the limbus at the 12-o'clock position developed into an abscess which opened spontaneously. Culture showed a pure growth of staphylococcus aureus. The nodule healed completely without ectasia of the sclera or any choroidal involvement. Edna M. Reynolds.

Wilson, R. P. **Lipoid degeneration of cornea and palpebral conjunctiva.** Giza Mem. Ophth. Lab., 1938, 13th rept., p. 81.

The tarsal conjunctiva of each eye was studded with small yellow dots beneath the epithelium and interspaced

with dilated capillaries. Both corneas presented a similar appearance. The overlying epithelium was smooth. Paraffin sections showed a diffuse infiltration with mobile histiocytes loaded with cholesterol. (4 drawings.)

Edna M. Reynolds.

Wong, R. T. **Band-shaped opacity of the cornea associated with juvenile atrophic arthritis.** Arch. of Ophth., 1941, v. 26, July, pp. 21-24.

Wong reports a case of bilateral band-shaped opacity of the cornea associated with chronic arthritis in a 13-year-old boy. The combination of the degenerative change of the cornea with chronic arthritis in children is regarded by some authors as a definite syndrome. This type of corneal dystrophy occurs in comparatively healthy eyes. A disturbance in nutrition with the resulting diminution of the ability of the cornea to withstand external injurious influences is considered an essential factor. In the early stages there is deposition of amyloid or hyaline degeneration. Later there are deposits of phosphates and carbonates of lime and of small quantities of sulphur and silicon. (Photograph.)

J. Hewitt Judd.

7

UVEAL TRACT, SYMPATHETIC DISEASE, AND AQUEOUS HUMOR

Ford, F. R., Walsh, F. B., and King, A. **Clinical observations on the pupillary phenomena resulting from regeneration of the third nerve, with especial reference to the Argyll Robertson pupil.** Bull. Johns Hopkins Hosp., 1941, v. 68, April, p. 309.

Five cases are reported illustrating disorders of ocular motility (mass movement) following regeneration of previously paralyzed third nerves. The accepted theory for these disorders is that the regenerating nerve fibers become misdirected. Especial reference is made to the occurrence of the Argyll Robertson pupillary reaction, which is explained on the same basis.

Robert O. Sherwood.

Irvine, R., and Irvine, M. B. **A study of aqueous humor as an aid to understanding inflammatory conditions of the eye.** Trans. Pacific Coast Oto-Ophth. Soc., 1940, v. 25, pp. 135-151.

The Irvines aspirated the aqueous and made various chemical determinations on 44 cases of intraocular disease, including uveitis, glaucoma, and separated retina, and on 12 rabbits with experimentally produced sclerokeratitis. The methods for obtaining and analyzing the aqueous are discussed. The sugar content of the aqueous varies primarily with the blood-sugar level. It is not affected by the etiology of the pathologic process but tends to be lower in cases with acute inflammation. The cell response is affected primarily by the acuteness and intenseness of the inflammation. Synechias vary in degree with the coincident increase in protein content of the aqueous and the length of time it has been increased.

Lawrence G. Dunlap.

Krotkov, H. V. **Adie's syndrome.** Viestnik Opht., 1941, v. 18, pt. 2, p. 207.

A young woman with negative serologic findings in the blood and cerebrospinal fluid, developed an inconstant mydriasis of the right pupil, with slow reactions to light and accommodation. She had in addition an inequality in the tendon reflexes of the upper arms, enlargement of the thyroid, atrophy of the right thenar and hypothenar eminences, and increased perspiration. (Review of the literature.) Ray K. Daily.

Lowry, J. T. **Iris changes in syphilis.** Southwest Med., 1941, v. 25, Jan., p. 14.

The author claims that the iris in patients with syphilis presents the following deviations from the normal: (1) The inner zone is set on a deeper plane in relation to the outer zone. (2) The line of demarcation between the two zones is more jagged and is not so concentric with the pupillary margin. (3) The outer segment is irregularly pitted by small oval or pear-shaped depressions whose long axes are perpendicular to the pupillary margin. (4) In a moderately large percentage of such patients the iris pattern is almost entirely lost. These conditions have been found by the author in 85 percent of all positive cases. In this work 376 negative and positive patients were examined.

Ralph W. Danielson.

Rubino, A. **Uveomeningitic syndrome.** Ophthalmologica, 1941, v. 101, June, p. 321.

The author describes a typical case of Harada's syndrome, the first one to be observed in Italy. He believes that the cause is not tuberculosis but a virus which is characterized by specific uveoneurotropism or uveomeningotropism. He ascribes to melanotropism of the virus the changes in hair and skin seen in the disease, and denies an endocrine or anaphylactic pathogenesis. (Incidentally, as a writer in "Nature" has pointed out, "melanodesmic" is more accurate than "melanotropic" and is not misleading.)

F. Herbert Haessler.

Satanowsky, Paulina. **Iridocyclitis with hypopyon and glaucoma.** Arch. de Oft. de Buenos Aires, 1939, v. 14, Dec., p. 1024.

Two cases of iridocyclitis complicated with hypopyon and glaucoma are reported. A very thorough survey failed to determine the etiologic factor. The author emphasizes the advantages of atropine and adrenalin in the treatment of these cases as well as iontophoresis with a weak solution of glaucosan.

Plinio Montalván.

Suárez Gabard, Santiago. **Syphilis and tonic pupil.** Arch de Oft. de Buenos Aires, 1939, v. 14, Dec., p. 1006.

A unilateral case of tonic pupil is reported in a woman of 55 years of age. There was anisocoria and the tonic pupil was the larger. In the affected eye the photomotor reflex was abolished, and the accommodation-convergence and Pilz-Westphal reflexes were tonic in character.

Neurologic examination revealed a positive Romberg with absence of tendon reflexes. A diagnosis of tabes was made from the neurologic standpoint. The blood Wassermann was persistently negative, but the family and past history as well as the clinical manifestations bore out the diagnosis of latent syphilis.

Plinio Montalván.

8

GLAUCOMA AND OCULAR TENSION

Clarke, S. T. **Gonioscopic observations in cyclodialysis operations.** Amer. Jour. Ophth., 1941, v. 24, Sept., pp. 1026-1028.

Dashevskii, A. I. **The significance of adaptation studies in the early diagnosis of prodromal glaucoma.** Viestnik Opht., 1941, v. 18, pt. 3, p. 241.

The author adds a new test for prodromal glaucoma—the pilocarpine-adaptation test. It is based on improved light sensitivity of glaucomatous eyes following instillation of pilocarpine. The adaptation curve is taken with an

artificial pupil, before and after instillation of pilocarpine. Better adaptation after pilocarpine instillation is diagnostic of glaucoma. Ray K. Daily.

Ehrlich, L. H. **Bilateral glaucoma associated with unilateral nevus flammeus.** Arch. of Ophth., 1941, v. 25, June, pp. 1002-1006.

A review of the literature revealed 96 cases in which nevus flammeus of the face had occurred with glaucoma, usually of the infantile type. Only one previous case was found in which ocular involvement was bilateral while the nevus was unilateral. The author reports the case of a lady, aged 24 years, in which this occurred. The symptoms of the syndrome and the cause and treatment of the ocular aspect are briefly discussed. J. Hewitt Judd.

Grigorieva, H. I. **Elastotonometric investigations in glaucoma.** Viestnik Opht., 1941, v. 18, pt. 1, p. 64.

The results of this investigation show a difference in the amplitude and level of the curves between normal and glaucomatous eyes. Pilocarpine changes the character of the elastotonometric curve, and four daily instillations are more effective than two. In 35 patients the elastotonometric findings conformed to the tonometric. In three cases the elastotonometric curve was pathologic, while the tonometric findings were normal. There is no correlation between the amplitude of elastotonometry and the height of intraocular tension. Elastotonometry has to be done with care, in order to avoid corneal erosions. Ray K. Daily.

Henderson, Thomas. **The mechanism of the intraocular pressure in mammalia.** Brit. Jour. Ophth., 1941, v. 25, Aug., pp. 349-356.

Henderson maintains that the simplest method of determining intraocular pressure is ophthalmoscopic examination. Experimental intraocular pressure, problems arising from the alleged high physiologic level of intraocular pressure, physiologic equilibrium between intraocular and intracranial pressures, and intraocular tension are the topical divisions under which the subject is discussed. Four deductions are made: (1) Clinical and physiologic problems cease to exist with the realization that the same mechanism and the same level of pressure obtain in the eye as in the brain; (2) Reaction of the central retinal vein at the disc to the least touch on the lid is the key to the understanding of the mechanism under discussion; (3) As in the brain, intraocular pressure is not dependent on volume but carries on and fluctuates with venous exit pressure; (4) The intraocular and the intracranial pressures are governed by the general circulatory pressure on the venous side. (Figures, references.)

D. F. Harbridge.

Hruby, K. **The mechanism of eserine and pilocarpine.** Graefe's Arch., 1940, v. 141, pts. 4 and 5, pp. 517-537.

Four conditions which may explain the lowering of intraocular pressure by miotics are discussed in detail: (1) the increase of iris surface; (2) the freeing of the angle; (3) alteration of the blood volume by effect on the vessels; (4) contraction of the ciliary muscle. The author found in his 281 cases of primary glaucoma that persons with hypoactive ciliary muscles (myopes) were affected earliest, while those with hyperactive ciliary muscles (hyperopes) were affected latest, and emmetropes were intermediate. The decreased activity of the ciliary muscle with age as

indicated by the decreased accommodative amplitude was considered to be the precipitating factor in producing primary glaucoma. The pressure-reducing effect of eserine and pilocarpine was therefore inferred to be due to their action on the ciliary muscle. As evidence the author points out that hyperopes reacted better than myopes to these drugs. Frances C. Cogan.

Irvine, Rodman. **Exfoliation of the lens capsule (glaucoma capsulare).** Arch. of Ophth., 1941, v. 25, June, pp. 992-1001. (See Section 9, Crystalline lens.)

Satanowsky, Pauline. **Iridocyclitis with hypopyon and glaucoma.** Arch. de Oft. de Buenos Aires, 1939, v. 14, Dec., p. 1024. (See Section 7, Uveal tract, sympathetic disease, and aqueous humor.)

Kalkutina, M. L. **The effect of pilocarpine on light sensitivity of the glaucomatous eye.** Viestnik Opht., 1941, v. 18, pt. 3, p. 250.

The instillation of pilocarpine in a glaucomatous eye produces improvement in light sensitivity, which is followed by a second phase during which light sensitivity falls again. By properly spaced instillations of pilocarpine the secondary phase can be eliminated, and the eye maintained in its optimum functional state, which probably corresponds to its optimum physiologic state. The pilocarpine adaptation test can thus be used clinically to determine therapeutic effectiveness.

Ray K. Daily.

Kaminskaja-Pavlova, Z. H. **Biomicroscopy of the anterior ocular segment in glaucoma, and the choice of surgical procedure.** Viestnik Opht., 1940, v. 18, pt. 2, p. 123.

One hundred eyes of sixty patients were subjected to biomicroscopy, with the objective of determining if biomicroscopic findings could be correlated to surgical procedures. In 36 cases there was decided dilatation of the anterior ciliary veins. Of 54 eyes with chronic inflammatory glaucoma, in 22 the anterior chamber was shallow, and in 32 it was of normal depth or deeper. Of 17 cases of acute glaucoma the anterior chamber was deep in 11, and normal or deeper in the rest. Of 16 cases of simple glaucoma the anterior chamber was deep in 5 and normal in 11. Among the sixty patients were 3 cases of anterior hemorrhagic glaucoma which is characterized by a deep anterior chamber, edema of the iris and dilatation of its vessels, absence of excavation, and an unfavorable reaction to pilocarpine. The depth of the anterior chamber is indicative of the location of the obstruction to drainage. If the interference with drainage is located in front of the lens the anterior chamber is normal in depth or even deeper. If the edema is behind the lens, the latter is pushed forward and the anterior chamber is shallow. The choice of surgical procedure is determined by the location of the obstruction to drainage. If the obstruction is in front of the lens, cyclodialysis or a filtering operation is indicated, except in acute glaucoma which calls for an iridectomy. In obstruction in the posterior chamber, cyclodialysis is ineffective unless associated with an iridodialysis which gives access to the posterior chamber. In cases of shallow anterior chamber with forward displacement of the lens, the disturbance is principally in the venous system and surgery should be undertaken cautiously because it is frequently ineffective. In such cases the most logical procedure is posterior trepana-

tion. Surgery of hemorrhagic glaucoma is frequently complicated by severe hemorrhages into the anterior chamber, which lead to loss of the eye.

Ray K. Daily.

Levina, L. S. **Nerve pathways for the regulation of intraocular tension.** Viestnik Opht., 1941, v. 18, pt. 1, p. 49.

The objective of this investigation was to demonstrate the nerve pathway for consensual ocular reactions, and it concerned itself with the bilateral rise in intraocular tension produced by irritation of one eye. This rise was produced by traumatism, by subconjunctival injections of 20-percent saline, and by tying the venae vorticosae. The rise in the tension of the nontraumatized eye was lower and occurred somewhat later. The pathogenesis of the involvement of the non-traumatized eye the author explains in the following manner: the pain in the traumatized eye produces a complex biologic reaction with vasodilatation and increased vascular permeability which leads to a rise in intraocular tension. The increased intraocular tension acts as an irritant to the inner receptors, which through afferent pathways to the higher nerve centers transmit the irritation to the second eye. Section of the ciliary nerves, which conduct pain sensation from the eye, does not block the development of increased tension in the second eye. Following a novocaine blockade of the ciliary ganglion, or extirpation of the superior cervical ganglion, the increased tension in the nontraumatized eye did not appear; which indicates that the impulse proceeds along these nerve pathways. This leads the author to the conclusion that the vegetative nervous system is the pathway for consensual paired ocular reactions. The failure of this reaction to develop under anesthesia indicates a participation of the cerebral cortex.

Ray K. Daily.

Popov, M. Z. **Types of sugar-tolerance curves and their clinical significance in glaucoma.** Viestnik Opht., 1941, v. 18, pt. 2, p. 161.

The objective of this investigation was to determine the type of blood-sugar-tolerance curve in glaucoma, as a reflection of the general neuroendocrine background predisposing to glaucoma; and to correlate the clinical course of glaucoma with the sugar-tolerance curves. The data show that glaucoma is developed in a disturbed hormono-vegetative environment, which exercises its destructive action on organs of least resistance. The sugar curves indicative of hyperirritability of the entire hormono-vegetative system were found in severe inflammatory glaucoma with high tension, rapid loss of vision, headache, ineffectiveness of therapy, and a history of haloes. This justifies the assumption that the basis of the inflammatory glaucoma is a hyperirritability of the sympathico-adrenal system, with corresponding hyperirritability of the superior cervical ganglion; the latter is intimately connected with the hypothalamus and the ciliary ganglion. The clinical symptoms are due to circulatory disturbances, principally in the choroid and brain. In simple glaucoma the sugar curves indicate a disturbance of the parasympathico-insulin system and liver, not so intimately connected with the vascular system as the sympathetic system. In this type of glaucoma the destruction is centered on the optic nerve; the author believes that the underlying factor in this process is auto-intoxication, resulting from a disturbed insulin metabolism. Further investigation in this field will lead,

the author hopes, to etiologic therapy of glaucoma. Ray K. Daily.

Sag, H. O. **Combination of retinitis pigmentosa and glaucoma.** Viestnik Opht., 1941, v. 18, pt. 3, p. 272. (See Section 10, Retina and vitreous.)

Tichomirov, P. E. **The size of the blind spot in glaucomatous and normal eyes, in the water-drinking test.** Viestnik Opht., 1941, v. 18, pt. 1, p. 57.

Enlargement of the blind spot in glaucomatous eyes after drinking a large quantity of water is attributed by the author to peripapillary edema, developed as a result of increased capillary permeability. In response to the test the blind spot of normal eyes remained unaffected, while in glaucomatous eyes it became enlarged within three hours. In operated eyes the blind spot enlarged in response to the test although the tension was lowered, indicating that the surgical procedure does not restore normal capillary permeability. Ray K. Daily.

Wiener, Meyer. **The aftercare of cataract and glaucoma, including their complications.** Trans. Pacific Coast Oto-Ophth. Soc., 1940, v. 25, pp. 124-134. (See Section 9, Crystalline lens.)

9

CRYSTALLINE LENS

Bellows, J. G., and Chinn, H. **Biochemistry of the lens. 15. Studies on the swelling of the isolated lens.** Amer. Jour. Ophth., 1941, v. 24, Sept., pp. 979-999.

Beltukova, A. A. **Two unusual cases of familial hereditary cataract.** Viestnik Opht., 1941, v. 18, pt. 3, p. 321.

A woman 39 years old, with a history of congenital cataracts, brought two children with cataracts to the clinic. In both the lenticular pictures were the same. In the center of the lens was a gray sphere bordered by glistening white projections, extending backward from the anterior pole of the sphere. The rest of the lens was cloudy. No etiologic factor could be demonstrated. (Illustrations.) Ray K. Daily.

Bloch, Fritz. **Replacement of the fixation forceps in operation for cataract by three rectus muscle bridle sutures.** Arch. of Ophth., 1941, v. 25, June, pp. 1035-1037.

The author states that the routine use of bridle sutures through the lateral and superior rectus muscles in cataract operations gives firmer fixation of the globe, less injury to friable conjunctiva, greater freedom in the use of the knife and the scissors in corneal sections, and better control of intraocular tension and the vitreous body.

J. Hewitt Judd.

Gapeev, P. I. **The vitamin-C content in the aqueous, lens, and blood, in cataractous and normal eyes.** Viestnik Opht., 1941, v. 18, pt. 2, p. 154.

A review of the literature and a tabulated report of the author's findings in 48 cataractous and 8 normal eyes. The material consisted of the aqueous and lenses removed in cataract extraction, and of eyes enucleated because of glaucoma or traumatization. The data show that the normal aqueous and lens contain a relatively large amount of vitamin C. The vitamin-C content of the aqueous and lens in cataract is somewhat lower than in normal eyes. The blood of normal patients has a higher vitamin-C content than the blood of cataract patients. The comparatively high content of vitamin C in the aqueous and lens of cataract eyes,

in the face of the reduced blood content, does not justify any conclusions relative to the etiologic significance of vitamin C. Ray K. Daily.

Irvine, Rodman. **Exfoliation of the lens capsule (glaucoma capsulare).** Arch. of Ophth., 1941, v. 25, June, pp. 992-1001.

Two cases of exfoliation of the lens capsule are reported in detail. In the first there was a spontaneously dislocated lens with marked involvement of the zonule in the exfoliating process. This observation and the microscopic sections of the lens indicate that exfoliation of the lens is a disease of the zonule as well as of the capsule (which suggests that the zonular-lamellar layer of the capsule is similar in structure to the zonule) and that the zonule is more friable in this condition, altering the steadfastness of the lens. The development of absolute glaucoma further indicates that the prognosis of any concomitant condition, such as glaucoma or cataract, is less favorable when exfoliation is present. In the second case there developed typical exfoliation of the lens capsule after irradiation of the orbit for tumor. This case is the first reported in which typical exfoliation of the lens capsule was seen after roentgen irradiation. The changes are shown in photographs and photomicrographs. J. Hewitt Judd.

Krasnov, M. L. **Two clinical case reports: arachnodactylia and bilateral dislocation of the lens; and conjunctival cyst after enucleation.** Viestnik Opht., 1941, v. 18, pt. 2, p. 198.

A woman thirty years old came with dislocation of the left lens into the anterior chamber and secondary glaucoma. Her infant had struck her in the eye six weeks previously. On examination she was found to have signs of arachnodactylia and dislocation of the right lens down and in. The left lens, which was uneventfully extracted, was of normal size.

In another case an inclusion cyst, the size of a hazel nut, developed in the conjunctiva 2½ years after an enucleation, and was treated by electrocoagulation. To avoid such occurrence the lips of the wound should not be inverted in the introduction of conjunctival sutures. Ray K. Daily.

Platova, Z. I. **A case of unusual congenital lenticular abnormality.** Viestnik Opht., 1941, v. 18, pt. 2, p. 200.

A description of a rare bilateral anomaly, in otherwise normal eyes, which consisted of one vesicle located subcapsularly under the anterior pole of the lens. The physiologic shagreen of the lens, which is disturbed in anterior polar cataract, was normal in this case, as was the anterior lens capsule.
Ray K. Daily.

Seif, H. A. **Subconjunctival dislocation of lens with prolapse of ciliary body.** Giza Mem. Ophth., Lab., 1938, 13th rept., p. 55.

A fleshy mass covering part of the cornea was excised and found to contain remains of a dislocated lens and a piece of prolapsed ciliary body. The inflammatory reaction surrounding the lens material was much greater than that surrounding the ciliary-body tissue. Edna M. Reynolds.

Urbanek, J. **Measures for prevention of complications during and after intracapsular extraction of cataract.** Klin. M. f. Augenh., 1941, v. 106, Feb., pp. 129-159.

Thorough examination of the patient before surgery is recommended. In

cases of high blood pressure, venesection of up to 400 c.c. of blood is made. Giving high dosages of vitamin C before and during the first days after operation is designed to avoid postoperative hemorrhage. To prevent pulling up of the pupil after operation, peripheral iridectomy is performed. In the author's opinion intumescent cataract is the only form of cataract where intracapsular extraction is not indicated. (2 tables, 2 curves.)

Gertrude S. Hausmann.

Wiener, Meyer. **The aftercare of cataract and glaucoma, including their complications.** Trans. Pacific Coast Oto-Ophth. Soc., 1940, v. 25, pp. 124-134.

Wiener reiterates the clear-cut postoperative directions for the aftercare of cataract and glaucoma cases and their complications as outlined in his recent book, giving logical reasons for each thing he does or does not do, or allows the patient to do. The iris-inclusion operation is recommended for postoperative glaucoma after cataract extraction. The author stresses the disastrous results of blood and cortex remaining in the anterior chamber although either one alone is ordinarily innocuous. The usual postoperative complications are dealt with thoroughly. Lawrence G. Dunlap.

Wilson, R. P. **Dislocated lens adherent to optic disc.** Giza Mem. Ophth. Lab., 1938, 13th rept., p. 59.

A case report.

Wolfe, Otis, and Wolfe, R. M. **Removal of soft cataract by suction.** Arch. of Ophth., 1941, v. 26, July, pp. 127-128.

A combined irrigating and suction tip is advocated for extraction of soft cataract. Fluid is injected through a narrow keratome incision into the anterior chamber through the smaller barrel of a double-barreled needle, while oral suction is maintained through the second, or larger, barrel. The construction of the apparatus is shown in drawings. J. Hewitt Judd.

10

RETINA AND VITREOUS

Baevskii, I. L. **Etiology and treatment of functional hemeralopia.** Viestnik Opht., 1941, v. 18, pt. 3, p. 261.

A review of the literature and a report of the results of carotin therapy in night blindness. The conclusions are that functional hemeralopia is a result of A-avitaminosis on a background of faulty diet, somatic disturbance, and unfavorable external environment; and that injections of provitamin A are effective in the treatment of this disturbance. Ray K. Daily.

Hallermann, Wilhelm. **The differential diagnosis of inflammatory and degenerative changes in the posterior pole of the eye.** Klin. M. f. Augenh., 1941, v. 106, March, pp. 337-339.

Six cases are reported with disseminated, small, sharply outlined, yellowish-gray spots, densest in the region of the macula. With the Gullstrand instrument the spots were found to be under the retina. Against a diagnosis of tuberculous choroiditis were the symmetric occurrence in both eyes and the minimal changes even after a prolonged period of time. (One illustration.)

Gertrude S. Hausmann.

Jess, Adolph. **The question of prophylactic retinal attachment in threatened separation.** Graefe's Arch., 1940, v. 141, pts. 4 and 5, pp. 538-544.

The author believes that persons with threatened separation of the retina

should be operated upon. Threatened separation is suggested when myopic or elderly persons complain of sudden clouding of vision, flashes of light, and floating opacities, especially persons with a history of previous separation in the other eye. Ophthalmoscopic evidence of threatened separation consists of localized thinning of the retina with or without hole formation, of pigmentary or fibrous changes in the retinal tissue, and of cyst formation. The author reports eight cases of threatened separation of the retina which were successfully operated upon by electrocoagulation. None of the patients had developed separation of the retina when last seen. The maximal follow-up time was 2½ years. Frances C. Cogan.

Loewenstein, Arnold. **Retinopathia centralis angiospastica (angioneurotica) and serosa allergica, and their relation to detachment of the retina.** Brit. Jour. Ophth., 1941, v. 25, Aug., pp. 369-383.

Four cases are reported. The first case showed the angioneurotic retinopathy to be of probably allergic origin. The second case was diagnosed as one of posterior scleritis with exudation both in the orbital tissues and subretinally, there being exophthalmos and limitation of eye movements with submacular or intramacular exudate. The third case showed a central fundus picture analogous to the second in connection with a typical tuberculous iridocyclitis. The fourth case showed a chemotic subconjunctival bleb with corresponding retinal detachment in a myopic eye. The allergic inflammatory reaction is fully described and identified. (Figures, references.)

D. F. Harbridge.

McKeown, H. S. **Retinal hemorrhages in the newborn.** Arch. of Ophth., 1941, v. 26, July, pp. 25-37. (See Amer. Jour. Ophth., 1941, v. 24, May, p. 598.)

Meves, Harald. **The origin of hemorrhages in the fundus in cases of subarachnoid hemorrhages.** Klin. M. f. Augenh., 1941, v. 106, March, pp. 339-342.

In a woman of 37 years large retinal hemorrhages occurred simultaneously with a subarachnoid hemorrhage. As anatomic examination of the eyes disclosed neither pathologic changes in the blood vessels nor a hematoma of the sheath of the nerve, the sudden increase in the pressure in the veins is assumed to have been caused by the increased pressure of the cerebrospinal fluid resulting from the intracranial hemorrhage. Gertrude S. Hausmann.

Meyer, F. W. **The histology of human and experimental tuberculosis with special emphasis on tuberculous retinitis.** Graefe's Arch., 1940, v. 141, pts. 4 and 5, pp. 408-499. (See Section 17, Systemic diseases and parasites.)

Morrison, W. H. **Osteitis deformans with angioid streaks.** Arch. of Ophth., 1941, v. 26, July, pp. 79-84.

The author summarizes eleven cases previously reported and adds a personal case of osteitis deformans with angioid streaks. In the latter case the retinal involvement was typical, extensive, and bilateral. The macula of each eye was just spared. Pseudoxanthoma elasticum was not present. Since subclinical osteitis deformans occurs frequently, patients with angioid streaks should be examined for this as well as for pseudoxanthoma elasticum. In Morrison's case, the normal phosphatase content of the serum indicated that the disease was quiescent. The angioid streaks are well illustrated by colored fundus photographs of each eye.

J. Hewitt Judd.

Sag, H. O. **Combination of retinitis pigmentosa and glaucoma.** Viestnik Opht., 1941, v. 18, pt. 3, p. 272.

The author reviews the literature, which contains 34 case reports of combination of these two grave ocular diseases in the same eye. He adds four cases to this number.

Ray K. Daily.

Samoilov, A. I. **The clinical significance and demonstration of retinal edema.** Viestnik Opht., 1941, v. 18, pt. 1, p. 17.

The author finds retinal edema widespread in a variety of ocular lesions, such as choroiditis, choked disc, rhinogenic neuritis, and glaucoma. The occurrence of retinal edema is due to the biologic and chemical processes in the retinal tissue, which respond with a similar reaction to a variety of irritants. The edema takes place chiefly in the region of the second neuron, and extends towards the neuroepithelium. Its presence is diagnosed by means of Bernstein's calcium-ionization test, examination of dark adaptation, and electric retinal sensitivity. Clinically the edema has diagnostic and prognostic significance. It indicates the presence of an early and active process, in a stage at which recovery is still possible; it calls for intense therapy, and contraindicates the use of edemagenic agents such as dionin, hypertonic salt solution, or diathermy. Ray K. Daily.

Stewart, C. P. **Nutritional factors in dark adaptation.** Edinburgh Med. Jour., 1941, v. 48, April, p. 217.

After a preliminary discussion of the mechanism of dark adaptation, the author notes some of the factors which influence this process. Vitamin-A intake is probably the most important factor, nevertheless, there has not been the close connection in many situations and experiments that one might expect. The rate diminishes in old age. Pupil diameter is important. Sex is without effect. Fatigue and lack of sleep appear to have an adverse effect on both the rate of dark adaptation and the power to see in dim light. In some cases of poor dark adaptation, the administration of vitamin C produced as much effect as increasing the vitamin-A intake. The mechanism is deficient in tuberculosis, gastric and duodenal ulcer, and minor infections. The relationship of dark adaptation to oxygen deficiency, to lack of vitamin B, and to carbohydrate metabolism is discussed. (44 references.) Ralph W. Danielson.

White, G., and Hassard, C. E. A. **Retinopathy in diabetics.** Canadian Med. Assoc. Jour., 1941, v. 44, June, p. 586.

One hundred diabetics at the Toronto Western Hospital were examined for fundus changes. It was found that the incidence of retinopathy increased with advancing age, duration and severity of the diabetes, and the association of cardiovascular-renal disease. Six cases of retinopathy were found without concurrent existence of cardiovascular-renal disease. The authors think that diabetic retinopathy is a definite entity in which the vessel walls are affected by some factor other than and in addition to those operating in cardiac disease, hypertension, or nephritis.

John C. Long.

11

OPTIC NERVE AND TOXIC AMBLYOPIAS

Dvorzetz, M. A. **Blood transfusion and retrobulbar atropine injections in optic-nerve lesions caused by wood al-**

cohol. Viestnik Opht., 1941, v. 18, pt. 2, p. 181.

The report of a case of wood-alcohol poisoning with total loss of vision, which recovered almost normal vision under treatment with two blood transfusions and ten retrobulbar atropine injections in addition to the usual diaphoretic, diuretic, and supportive measures. Ray K. Daily.

Gelfand, B. **The action of calcium and magnesium ions on edemagenic scotoma.** Viestnik Opht., 1941, v. 18, pt. 3, p. 268.

This investigation demonstrates that magnesium ions have the same effect as calcium ions on peripapillary edema, and that both diminish it. Clinically this effect is manifested by diminution in the size of the blind spot. The author believes that both metals act directly on the retinal tissue, and not through the vasomotor innervation of the retinal vessels. Ray K. Daily.

Kalkutina, M. L. **Dark adaptation in diseases of the optic nerve.** Viestnik Opht., 1941, v. 18, pt. 3, p. 254.

The data show that in optic atrophy light sensitivity is always lowered. In edema of the optic disc adaptation remains normal for a long time, while inflammatory processes always impair it. The dark-adaptation data may therefore serve to differentiate edematous from inflammatory processes.

Ray K. Daily.

Kazas, I. I. **Therapy of luetic optic atrophy.** Viestnik Opht., 1941, v. 8, pt. 2, p. 142.

The author uses a Soviet preparation similar to stovarsol. The administration of this arsenical is preceded by a preparatory phase of atropine administered internally, and pilocarpine instilled into the conjunctival sac.

Ray K. Daily.

12

VISUAL TRACTS AND CENTERS

Belloni, G. B., and Fasiani, G. M. **The traumatic syndrome of the third visual neuron.** Riv. Oto-Neuro-Oft., 1940, v. 17, Nov.-Dec., pp. 491-516.

Eight cases are reported of lesions of intracranial optic pathways following fracture of the skull. Four of the cases showed immediate symptoms, either chiasmatic or unilateral, and four showed late symptoms. In two of the latter the visual disturbances were bilateral and progressive. Seven of the cases were operated upon by the transfrontal intradural route. A description is given of the anatomic lesion found in each case. Only two of the patients regained partial visual power, while full vision was regained by a girl of five years affected by a cyst of the chiasmatic region. The article is closed with a discussion on the different forms of arachnoiditis following trauma, and on the pathology of immediate and late forms of optic-pathway lesions and their surgical treatment.

M. Lombardo.

Bushpan, A. **A case of puerperal amaurosis.** Viestnik Opht., 1941, v. 18, pt. 3, p. 325.

A report of a case of functional blindness without ophthalmoscopic changes and with normal pupillary reactions in a woman twenty years old, immediately following delivery. Recovery occurred in ten days. Ray K. Daily.

Klemme, R. M. **Oculogyric crises.** Amer. Jour. Ophth., 1941, v. 24, Sept., pp. 1000-1004.

Lebensohn, J. E. **Wollaston and hemianopsia.** Amer. Jour. Ophth., 1941, v. 24, Sept., pp. 1053-1056.

Lisch, Karl. **Cerebral metamorphopsia.** Graefe's Arch., 1940, v. 141, pts. 4 and 5, pp. 555-558.

Two cases of cerebral metamorphopsia are reported in patients who had had nonperforating head injuries sometime previously. The first patient had a homonymous inferior hemianopsia. He observed that vertical lines and circles were distorted symmetrically but in opposite directions for each eye. The distortion was greatest in the lower half of the field. The second patient had an almost complete right homonymous hemianopsia with some overlapping of the left field below. This patient noted that vertical lines appeared to be rotated counterclockwise for each eye. Circles showed a corresponding distortion. These findings support Best's theory that cerebral metamorphopsia is due to an illusion caused by the visual defect rather than to a cyclophoria as assumed by Pötzl, or to a displacement of the visual cells in the occipital cortex as suggested by Lenz.

Frances C. Cogan.

13

EYEBALL AND ORBIT

Dodds, G. E. **Tuberculoma of the eye.** Brit. Jour. Ophth., 1941, v. 25, Aug., pp. 384-387.

In a 12-year-old Yoruba boy the disease had reached an advanced stage infrequently seen in Europe in this day. When brought to the eye clinic the boy had spinal caries with spastic paralysis of the lower extremities, and early death was thought to be imminent. Two years previously there had been pain in the back followed by swelling and paralysis of the legs. The left eye presented a tuberculoma. The globe was enucleated after three months of general treatment, during which time the patient's condition had improved and the tuberculoma had diminished in size. The pathologic condition of the eye is described. (Figures.)

D. F. Harbridge.

McNair, S. S. **Ludwig's angina complicated by orbital cellulitis.** Arch. of Ophth., 1941, v. 25, June, pp. 1033-1035.

The case reported is that of a woman aged 49 years, in whom Ludwig's angina complicated by orbital cellulitis developed four days after the removal of a lower first-molar tooth.

J. Hewitt Judd.

Wilson, R. P. **Bilateral inflammatory pseudotumor of the orbit.** Giza Mem. Ophth. Lab., 1938, 13th rept., p. 86.

Hard swellings arising within the orbital cavities, and pushing the upper lids forward proved to be chronic inflammatory pseudotumors in a stage of advanced fibrosis. After local short-wave therapy given twice weekly for 15 weeks, the swellings were reduced to about one half their original size and assumed a bony hardness. There was at no time any limitation of movement of either eye and no diplopia.

Edna M. Reynolds.

NEWS ITEMS

Edited by Dr. Ralph H. Miller
803 Carew Tower, Cincinnati

News items should reach the editor by the twelfth of the month

Deaths

Dr. Olin Everett Farley, New York, New York, died August 5, 1941, aged 49 years.

Dr. Irving D. Cole, Clarksburg, West Virginia, died August 10, 1941, aged 60 years.

Dr. John Raymond Sackrider, Santa Barbara, California, died August 4, 1941, aged 63 years.

Dr. Oliver Fletcher Best, Providence, Rhode Island, died in August, 1941, aged 79 years.

Dr. George Maitland Biggs, Toronto, Canada, died August 25, 1941, aged 60 years.

Dr. George Abram Zimmerman, Harrisburg, Pennsylvania, died August 15, 1941, aged 61 years.

Dr. Francis Wayles Shine, Charlottesville, Virginia, died September 24, 1941, aged 67 years.

Dr. William A. Ellingwood, Rockland, Maine, died August 30, 1941, aged 58 years.

Dr. Joseph Morris Rosenthal, Philadelphia, Pennsylvania, died September 3, 1941, aged 56 years.

Miscellaneous

In the August issue of the British Journal of Ophthalmology it was announced that the sixth volume of Neighborhood Health has been devoted to sight conservation. Articles are included by Dr. Rice, Dr. Berens, Miss Merrill, Dr. Kirby, and Dr. Thygeson on the subjects of the health department in the prevention of blindness, advances in eye surgery, the story of eye glasses, syphilis of the eye, and gonococcal infections of the eye, respectively. There are additional articles of interest and columns of questions and answers of practical appeal.

The seventh semi-annual postgraduate course in neuromuscular anomalies of the eyes was given at the Children's Memorial Hospital, Chicago, November 9th to 14th under the direction of Dr. George P. Guibor.

The Manhattan Eye, Ear, and Throat Hospital announces the following series of evening lectures given as graduate instruction to the resident staff in the department of ophthalmology: "The prescription of glasses in presbyopia" by Dr. W. Hipp; "Orbital infections" by Dr. L. Hubert; "Developmental anomalies of the eye" by Dr. P. Montalván; "Ocular tuberculosis" by Dr. R. T. Paton; "Heredodegenerative diseases of the eye" by Dr. M. A. Last; "Ocular malingering in relation to the selective service act" by Dr. G. Frey; "Corneal dystrophies" by Dr. G. Bonaccolto; "Keratectomies and keratoplasties" with motion pictures by Dr. R. Castroviejo; "War injuries of the eye" by Dr. M. A. Last; "Diseases of the lacrimal apparatus" by Dr. P. Montalván; "Ocular syphilis" by Dr. R. T. Paton; and "Diagnostic and therapeutic use of tuberculin in ophthalmology" by Dr. A. A. Eggston.

Societies

The Puerto Rico Medical Association has announced that it has recently organized a Section of Ophthalmology and Otorhinolaryngology with the object of promoting scientific advancement of these branches of medicine in the island.

At the meeting of the Dallas Southern Clinical Society, October 27th to 29th, Dr. Dan Brannin discussed the subject, "The eyes in diabetes" at a symposium covering the general problems of diabetes mellitus.

Personals

Dr. Henry P. Wagener, Rochester, Minnesota, was the guest of honor at the Kansas City Southwest Clinical Society and the Kansas City Society of Ophthalmology and Otolaryngology on October 9, 1941. He conducted an eye clinic and read two papers, one on "Ocular lesions in vitamin deficiencies" and another on "The grading and grouping of hypertensive lesions in the retina."

Dr. Alson E. Braley, of Detroit, has been appointed assistant professor of ophthalmology at the College of Physicians and Surgeons, Columbia University.

On Wednesday, October 27th, an informal breakfast, given by Dr. Everett Gore, of Houston, was held in honor of Sir Harold Gillies and Dr. Perry Goldsmith, guests of the American Academy of Ophthalmology and Otolaryngology, at the Palmer House, Chicago.

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Rules for Glaucoma Patients

1. Carefully follow your eye physician's instructions and remember especially to return for reexamination at the appointed time.
2. Consult him at once if you see rainbow-colored halos around lights, if the eye becomes painful, or the vision is blurred, or sight impaired in any way at all.
3. Avoid as much as possible excitement, anger, worry, fear or disappointment.
4. Take care that bowel movements are regular.
5. Avoid tight-fitting clothes—a tight collar, corset, or belt.
6. Keep your blood circulation active. If occupation compels you to sit the entire day, take a long but not too tiring walk before and after work.
7. Keep your teeth clean and healthy; pay attention to acute or chronic colds.
8. Limit drinking coffee and tea (not too strong) to one cup a day. Avoid alcoholic drinks.
9. Keep bedroom well ventilated and at a moderate temperature; around 70 degrees (Fahrenheit).
10. Avoid dark rooms as much as possible. Go to movies only if your eye physician permits. Remain at the movies for only one feature and, if possible, choose subjects that are not depressing or upsetting for you.
11. Do not use any drops or eye washes without consulting your eye physician. They may be very harmful.
12. Have a periodic (yearly) examination of your entire body by your family physician.

Committee on Glaucoma
National Society for the Prevention of Blindness

CRITICISMS, COMMENTS OR SUGGESTIONS ARE WELCOME

AUTHOR INDEX

Compiled by EMMA S. BUSS

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Abbott, H. B. (959).
Adam, C. (359).
Adamantiadis, B., and Djacos, C. (227).
Adler, F. H., and McDonald, Robb. 77.
  and Scheie, H. G. (595).
  and see McDonald, R.
Adrogué, Esteban. (220), (348), (1091).
  and Cammarota, A. (969).
Agatston, S. A. 570.
Ageloff, Harry. (224).
Agnello, Francesco. (344), (1093).
Agundis, Teodulo. (1228).
Aird, R. B. (476), (834).
Aires, Francisco. (1224), (1341).
Ajo, C. (1341).
Alagna, Gaspare. (1329).
Albaugh, C. H. **690.**
Albers, E. C. (1097).
Alexander, G. F. (1215).
Allen, F., and Schwartz, M. (220).
Allen, J. H., and Barer, C. G. (473).
  and see O'Brien, C. S.
Allman, Lieutenant. 1441.
Alvaro, M. E. (605), (1219).
Amsler, Marc. (1085).
Andrade, Cesario de. (1220).
Andresen, E. E. (722).
Angevine, D. M., and Huntington, R. W., Jr. (1099).
Angius, Tullio. (1228), (1330).
Anglani, Alceste. (239).
Anquin, M. H. de, see Zavalia, U. A.
Appelbaum, Alfred. (360).
Appelmans, M. (89).
Apple, Carl. 697, 697.
Archer-Hall, H. W. (841).
Arcuri, Domenico. (1212).
Arndt, E. W. (1228).
Asarova, H. C. (1447).
Ascher, K. W. **615.**
Askalonova, T. A., and Vilovskaja, K. E. (1213).
Aten, A. H. (91).
Athens, A. G. **1138.**
Aurand. (353).
Ayres, Francisco. (1330).
Azerad, E., see Terrien, F.

Bab, Werner. **759.**
Babel, J. (1076).
Bachman, W., see Meesmann, A.
Badtke, Günther. (462), (478).
Baevskii, I. L. (1468).
Bahn, C. A. **1309.**
Bahr, Gunnor. (236).
Bailey, John. 567.
Bailliart, P. (965).
  and Bellot, A., and Tillé, H. (838).
  and Schiff-Wertheimer, and Juvanon, L. (239).
Baird, M., and Clay, G. E. (91).
Bakarenkov, B. T. (601).
Baker, J. P. (714).
Bakker, A. (111), (111), (1086), (1337).
Balaco, Franco. (107).
Balding, L. G. (108).
Ballantyne, A. J. (970), (1341).
Baltin, M. M., and Sviadocz, B. I. (1338).
Balza, J., see Cramer, F. E. K.
Bamford, C. H., and Barber, H. (1090).
Bane, W. M. 329.
  and Sherwood, Robert. 701.
  and Weston, Horace. 702, 702.
Bangerter, A. (975), (1097).
Bannon, R. E., see Ogle, K. N.
Baquis, Mario. (1338).
Barbel, I. E., and Benin, P. H. (960).
Barber, H., see Bamford, C. H.
Barbour, F. A., and Fralick, F. B. **553.**
Bardanzellu, Tomaso. (714).
Bardelli, L., see Jeandelize, P.
Barer, C. G., see Allen, J. H.
Barkan, Otto. (215), (234), **439, 768.**
Barkhash, S. A. (1332).
Barnshaw, H. D., and Read, W. T., Jr. (89).
Barsoum, Fahim. (1454).
Bartels, M. (223).
  and Loens, M. (1224).
Bartley, S. H. 1446.
Basile, Giambattista. (237).
  and see Nastri, F.
Battignani, Agostino. (95), (355).
Baudouin, A., Halbron, P., Fischgold, H., and Mion, R. Y. (215).
Bauer, Carlos. (603).
Bazshenova, M., see Filatov, V. P.
Beach, S. J., and Holt, E. E., Jr. **668,** (970).
Beard, Hallard. 694.
Beau, A., and Cordier, J. (847).
Beliakov, A. A. (462).
Belloni, G. B., and Fasiani, G. M. (1471).
Bellot, A., see Bailliart, P.
Bellows, J. G., and Chinn, H. (469), (957), (958), **979,** (1083).
Belousova, P. V. (1097).
Beltukova, A. A. (1466).
Bender, M. B., and Kanzer, M. G. (1091).
Benedict, W. L. (472), **1005.**
  and see Parkhill, E. M.
Benin, P. H., see Barbel, I. E.
Benstein, I. I. (217).
Berens, Conrad. **66,** (221), (221), (221), **689, 804,** (846), **915,** 1072, (1101), **1192.**
  and Elliot, A. J., and Sobacke, Lucille. **1418.**
  and Nilson, E. L. **645,** (969).
  and Rothbard, Sidney. **550.**
Berezinskaja, D. I. (459), (969).
Berkovich, M. E. (605).
Berliner, M. L., and Gartner, S. (349), 567.
Berrettini, G. L. (1213), (1343).
Bertoldi, Maria. (1222).
Bertotto, E. V. (716).
Bettman, J. W., and McNair, G. S. **1190.**
Beyer, W. (1448).
Bhaduri, B. N. (346).

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Bidyādhar, N. K. (1101).
Bietti, Giambattista. (227), (726).
Bikhovskii, M. A., and Kaganova, O. (600).
Bilit, M. B., see Sergievskii, L. I.
Biró, Imre. (101), (1094).
Blaauw, E. E. (970).
Black, N. M., Jr., see Leinfelder, P. J.
Blaess, M. J. (91), (473).
Blanc, E. (234).
Blanchard, E. L., and Harper, H. A. (714).
and see Jeans, P. C.
Blanchi, Guido. (1227).
Blankstein, S. S. **895.**
Bloch, Fritz. (1466).
Bloch, H. S., see Meyer, Karl.
Blonstein, M., see Levitt, J. M.
Bloom, W. A., Leech, M. P., and Shaw, W. J. (1329).
Blumenfeld, H. A. (1454).
Blumenthal, C. J. (101).
Böck, Joseph. (824).
Boehm, von Gundo. (460), (460).
Bogart, D. W. (1211).
Bogdanov, I. L. (1074).
Bogoslovski, A. I., and Rosenblum, M. E. (240).
Bolettieri, Daniele. (96), (1099).
Bolgob, P. I. (472).
Bonamour, G., see Bonnet, P.
Bonnet, Paul. (240), (240), (351), (353), (824), (841), (965).
and Bonamour, G. (224), (841), (842).
and Chauviré. (355), (970).
and Grandclément, E. (237).
and Paufique, L. (353).
Bonnet, R., see Puech, P.
Borezovskaja, S. (958).
Boros, Béla. (92).
Borsello, Giuseppe, and Brunetti, F. (1229).
Borsotti, Ippolitto, and Pillett, P. (360).
Boshoff, P. H., and Rethmeier, B. J. (104).
and see Dethmers, P.
Bossalino, G., and Sironi, L. (1213).
Bowen, Ralph. (1074).
Bowman, W. (605).
Bracken, Franklin. 70.
Braendstrup, Paul. (842).
Braley, A. E. (225), (962).
Brav, S. S., see Lehrfeld, Louis.
Brawley, F. T. 1209.
Breisacher, C. F., see Lehrfeld, Louis.
Brewster, H. F. (846).
Bristow, J. H. (217).
Brobeck, V. H. 329.
Broda, E. E., Goodeve, C. F., and Lythgoe, R. J. (221).
and Victor, E. (1215).
Brodskii, B. S. (1097).
and see Kalfa, C. F.
Brodsky, I., and Waddy, G. (107).
Brons, Hilari. (354).
Brooks, Roosevelt. 695, 696, 696.
Broun, G. O., see Griffith, W. L.
Brown, A. L. (92).
Brown, G. L., and Harvey, A. M. (1345).
Brown, R. W., see Watkins, C. H.
Brown, W. M. (1086).
and Whitney, E. L. (474).
Brückner, Roland. (596), (1220).
Bruens, Egon. (464).
Brunetti, F., see Borsello, G.
Bücklers, Max. (726).
Bucy, P. C., see Halstead, W. C.
Bujadoux, A. (836), (958).
Burch, E. P., see Stenstrom, W. K.
Bürki, E. (217), (1456).
Burky, E. L. 704.
and see Woods, A. C.
Burleson, J. H., and McCurdy, M. W. (1227).
Burmistrova, E. (1332).
Burri, Clara. (223).
and see Cushman, Beulah.
Bursuk, G. G. (1224).
and Schultz, V. A. (601).
Busacca, Archimède. (477), (975).
Buschke, Wilhelm. 69.
and see Friedenwald, J. S.
Bushmich, see Byshmich, D. G.
Bushpan, A. (1471).
Bussy, Juliette. (351).
Butler, T. Harrison. (1097).
Bycigin, A. (458).
Byshmich, D. G. (603).

Callahan, Alston. **196.**
Calvery, H. O., Lightbody, H. D., and Rones, B. (1213).
Cameron, Walter. **687.**
Cammarota, A., see Adrogué, Esteban.
Campailla, Giuseppe. (356).
Campbell, A. C. P., see Michaelson, I. C.
Campion, G. S. (216).
Carlberg, Olof. (838).
Carroll, F. D. (104), 331.
Carruth, H. E. (349).
Cashell, G. T. W. 808.
Cass, E. E. 808.
Castañe Decoud, D.D. de. (1222).
Casten, V. G. 200, 202, 202, 813.
Castroviejo, Ramón, **1, 139.**
Cauer, R. (1338).
Caveness, H. L., Satterfield, G. H., and Dann, W. J. (1211).
Cavka, Vladimir. (1457).
Cecchetto, Ezio. (1448).
Cerboni, F. C., see Lijo Pavia, J.
Chaffee, Eleanor, see Meyer, K.
Chamberlain, Webb, 448.
Chamberlain, W. P., Jr., see Elliot, A. J.
and see Meyer, Karl.
Chance, Burton. 78, **519,** (972).
Chand, Ratan. (240).
Chang, E., see Luo, T. H.
Charamis, J. S. (356), (459).
Charlin, C. C. 86, **1392.**
Charosky, L., see Steinberg, I. R.
Chauviré, see Bonnet, P.
Chavira, R. A. (597).
Chechik-Kunina, E. A. (592).
Chepurina, T. S. (1338).
Chernobilskaja, P., see Kholina, A.
Chetverikov, V. (1448).
Chinn, H., see Bellows, J. G.
Chisholm, J. F., Jr. (227), 949.
Chuprakov, A. T. (1215).
Chutko, M. B. (962).

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Cibis, Paul. (346), (1345).
Clarke, S. T. **1026.**
Clay, G. E., see Baird, M.
Cogan, D. G. (1076), (1216), **1431,** (1457).
Colditz, H. (1339).
Coles, J. D. W. A. (962).
Colson, Z. W. (599).
Cook, G. M., see Mitchell, H. S.
Cooke, C. T. (113).
Coombs, J. W. 695.
Cooper, E. L. (1084).
Cooper, W. L. (225).
Coppez, J. H. (96).
Coppola, Luigi. (462).
Corboy, P. M. **57,** 700.
Cordes, F. C. 818, **1057,** 1323.
Cordier, J., see Beau, A.
Corrado, Antonio. (1081).
Corser, J. B. (1224).
Cosgrove, K. W., and McCall, E. F. (1090).
Cosmettatos, G. F. (1084).
Cosserat, B. (830).
Costa Aguiar, Paulo da. (1333).
Cotlier, I. (599), (1216).
Coughlan, C. H., see Kluever, H. D.
Courtial, see Dollfus, E. J.
Courtney, R. H. 212, 1208.
and see Stone, J. B.
Covitz, E. E. **1423.**
Cowan, A. 442.
Cowan, T. H. 562, **1429.**
Cramer, F. E. K., and Balza, J. (478).
Cridland, Nigel. (1220).
Crisp, W. H. 82, 207, 337, 453, 580, 705, 816, 1064, 1204, (1227), 1321, 1443.
Cristini, Giuseppe. (1334), (1341).
Crozier, W. J., and Wolf, E. (1103), (1450).
Cruise, R. R. (971).
Csapody, István de. (218), (1094).
Culler, A. M. (601).
Cullom, M. M. **803.**
Cushman, Beulah, and Burri, Clara. **1044.**
Cusick, P. L. (102).
and Hawn, H. W. (1216).
Cutuly, E. (470).

Dahlsgaard-Nielsen, Esther. (231).
Dalen, Albin. (846).
Dammskaja, H. M. (598).
Dandy, W. E. (830), 1071.
and Follis, R. H., Jr. **365.**
Danielson, R. W., and Long, J. C. 330, **655.**
Dann, W. J., and Yarbrough, M. E. (1086).
and see Caveness, H. L.
Dashevskii, A. I. (234), (1339), (1462).
and Marmorstein, F. F. (356), (842).
Dashevskii, A. M. (975).
David, M., see Sourdille, G. P.
Davidov, V. G., see Samoilov, A. I.
Davids, Herman. (824).
Davidson, M. L. 70, 811.
Davis, L., and Martin, J. (1092).
Davis, W. T. (89).
Davson, H., and Quilliam, J. P. (96).
and see Duke-Elder, Sir W. S.
Dax, E. C. (595), (597), (1086).
Deans, S. R. (112).
Decoud, A. C., and Schujman, S. (1332).
DeCoursey, E., see Riwchun, M. H.
Dejean, C., and Roux, J. (720).
and Viallefont, H. (825).
De Long, Perce, see Fry, W. E.
Demole, V., and Knapp, P. (1100).
Denig, Rudolf. (234).
Dering, S. A. (1454).
Desvignes, Pierre, and Offret, G. (832).
and see Dollfus, E. J.
Dethmers, P., and Boshoff, P. H. (218).
Deutman, A. F. (92).
Dias, A. B. (1342).
Dieffenbach, Pierre, see Redslob, E.
Dinnean, F. L., see Stone, L. S.
Dischkov, S. A. (592).
Djacos, C., see Adamantiadis, B.
Dobson, Margaret. (719).
Dodds, G. E. (1472).
Doesschate, G. Ten. (114).
Doherty, W. B. **790,** (1092).
Dollfus, M.-A. (354).
Dollfuss, E. J., and Courtial, and Desvignes, P. (351).
Doyne, P. G. 806.
Drachman, M., and Friedman, B. (479).
Drews, L. C. **1403.**
Droeber, H. (836).
Dubois-Poulsen, A., see Magitôt, A.
Dudinov, O. A. (464), (1095).
Duggan, J. N. (346).
Duggan, W. F. (470), (831), (965).
Duke-Elder, Sir W. S. 585.
and Quilliam, J. C., and Davson, H. (96).
Duroux, see Talbot.
Duval, E., see Sourdille, G. P.
Duverger. (726).
Dvorzetz, G. A. (722), (1470).
Dzshanpoladova, A. C. (958).

Eames, T. H. **1170.**
East, T., and Savin, L. H. (464).
Eber, C. T. 942.
Eckhardt, R. E., and Johnson, L. V. (825).
and see Johnson, L. V.
Eggers, Harry. (1450).
Ehrlich, L. H. (1463).
Eidelman, B. M. (1216).
Elder, A. T. (107).
Elder, N. G. (1334).
Elles, N. B. (720), (1454).
Ellett, E. C. (477), (835).
Elliot, A. J. (89), **682,** 812.
and Chamberlain, W. P., Jr., and Givner, I. (966).
and see Berens, Conrad.
Ellis, R. W. B. (972).
Ellison, F. S., see Stone, L. S.
Elwyn, Herman. (347), (825).
Engel, Sam. **438,** (461).
Ershkovich, I. G. (1081).
Esaylova, Z. A. (598).
Evans, J. J. (356).
and Singer, E. (1447).
Evans, J. N. **507,** (714), (833), 1195, 1200, (1211).
Evans, W. H. (109).

Faber, E., and Roelofs, C. (114).

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Fanta, H. (240).
Farina, F. (1332).
Farina, P., see Fazio, C.
Farnarier, G., see Mastier, P.
Fasiani, G. M., see Belloni. G. B.
Fazakas, Alexander. (1224).
Fazio, C., and Farina, P. (475).
Federov, H. T., see Samoilov, A. I.
Feigenbaum, Aryeh. (360).
Feldman, J. B. (1451).
Feldman, M. F. (1074).
Ferrara, Aristide. (716).
Ferree, C. E., and Rand, G. (114), (458).
Ferrer, Horacio. **788.**
Fieandt, Olof. (1339).
Filatov, V. P., and Bazshenova, M. (1077).
and Volokitenko, A. (1217).
Filippi-Gabardi, Eugenio. (1452).
Fine, Max. (1457).
Fink, M. B. (357).
Fink, W. H. 703.
Fischer, F. P. (237).
Fischgold, H., see Baudouin, A.
Fisher, K. M. (1102).
Fishman, P. R., see Gleser, B. M.
Fletcher, D. E. (221).
Focosi, Marcello. (219).
Folk, M. R. (225), 697, 698, 698.
Follis, R. H., Jr., see Dandy, W. E.
Fontana, L. (1449).
Ford, F. R., Walsh, F. B., and King, A. (1461).
and see Walsh, F. B.
Fortin, E. P. (960), (1103).
Foss, Bjorn. (1449).
Fowler, M. J. 559.
Fralick, F. B., see Barbour, F. A.
Franks, A. G. (836).
Friede, R. (1458).
Friedenwald, J. S. 1201, 1202, 1203.
and Buschke, Wilhelm. **1105.**
Friedman, Benjamin. (96), (216), (222), (354), 441, **935,** (1075), (1087), (1097), (1211), (1448).
and Sallmann, L. von. 569.
and see Drachman, M.
Frocht, M., see McKinney, I. M.
Fronimopoulos, John. (715), (835).
Fry, W. E. 564, 1436.
and De Long, Perce. **664,** (1095).
Fuchs, A. (231).

Gabriélidès, C. A. (472).
Galan, R., see Schiff-Wertheimer, S.
Galeazzi, Cesare. (240).
Galfano, Antonino. (846).
Gallardo, Edward, see Thompson, Richard.
Gamble, R. C. **49.**
Gamet, J. H., see Smith, J. E.
Gandolfi, Carlo. (726), (1227).
Gans, J. A. 559, 560.
Gapeev, P. I. (1466).
García Querol. A., see Hurtault, J.
Gardner, W. J., and Shively, F. L., Jr. (97).
Garkavi, P. A. (1458).
Gartner, Samuel. (825).
and see Berliner, M. L.
Geilikman, O. B. (1339).
and Gitman C. M. (360).
Geis, Franz. (1330).
Gekhtman, M. I. (966).
Gekker, I. P. (963).
and Skvirskaja, Z. M. (465).
Gelfand, B. (1471).
Geller, I. M. (961).
Gendron, see Sourdille, G. P.
Genet, L. (219), (226), (237), (826), (837), (963), (966).
Georgiadés, G. (837).
Gerasimenko, T. H. (1087).
Gibson, G. G. 565.
and see Roesler, H.
Gifford, S. R. (462), (478), 1071, (1073).
and McPherson, William. 560.
Gilbert, G. P., see Luschinskii, G. F.
Gilbert, W. (1334).
Gill, W. D. **322.**
Gillan, R. U. (842).
Gillette, D. F. (1084).
Giqueaux, R. E. (1334).
Gitman, C. M., see Geilikman, O. B.
Givner, Isadore. (109), 446, 568.
and see Elliot, A. J.
Gleser, B. M., and Fishman, P. R. (604).
Gleserov, S. L. (600).
Godtfredsen, Erik. (833).
Goedbloed, J. (92).
and Wÿers, H. J. G. (1334).
Goldberg, F. P. (1221).
Goldberg, H. K., and Schlivek, K. (966).
Goldberg, Sol. **933.**
Goldenburg, M. (725).
Goldsmith, J. (1449).
Good, Palmer. (224), **794.**
Goodeve, C. F., see Broda, E. E.
Gordon, B. L. (846).
Gradle, H. S. 80, (115), (971), 1066.
Gralnick, Alexander. **26, 1174.**
Grancini, Enrico. (1092).
Grandclément, E., see Bonnet, P.
Grant, F. C., see Weinberger, L. M.
Graue y Glennie, Enrique. (465).
Graves, Basil. (592).
Greear, J. N., Jr. (604).
Green, A. S., and Green, M. I. (228).
Grawitz, P. B. (1458).
Green, M. L. 1438.
Greenwood, A. (115), 1438.
Griffith, W. L., and Broun, G. O. 1437.
Grignola, Antonio. (1080).
Grigoriev, H. I. (1221), (1463).
Grimm, R. (1452).
Grósz, Stephen de. (115), (216), (844).
Guglianetti, Luigi. (1343).
Guillaumat, L., see Puech, P.
Gundersen, Trygve. (459).
Gurvish, B. A. (1458).
Guy, Loren. 449.
Guyton, J. S. (222), **292.**
and Woods, A. C. **428.**

Hagedoorn, A. (92), (826).
Haig, C., see Patek, A. J., Jr.
Halbron, Pierre, see Baudouin, A.
Hallermann, Wilhelm. (1468).

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Halper, P. A. 560.
Halstead, W. C., Walker, A. E., and Bucy, P. C. (475), (833).
Hamilton, J. B. (592).
Hamman, G. C., see Holmer, W. J.
Handman, M. (351).
Hanono, M., see Levin, E.
Hansen, K., and Peters, E. (844).
Hanström, Erik. (116).
Harbart, Fred. (109).
Harman, N. B. (1450).
Harms, Herlyn. (224).
and Renner. (462).
Harper, H. A., see Blanchard, E. L.
Harrison, Colonel. 1440.
Harrison, W. J. **933.**
Hartinger, H. (222).
and Schubert, F. (716).
Hartmann, Edouard. (826).
Harvey, A. M., see Brown, G. L.
Hassard, C. E. A., see White, G.
Hausman, Louis. **119.**
Haviland, J. W., and Long, P. H. (112).
Hawn, H. W., see Cusick, P. L.
Hay, J. C., and Roaf, H. E. (222).
Hayden, Reynolds. (1102).
Heath, Parker. 701, (1084), **1433.**
and see Sachs, Erich.
Heglo, L. G. (1217).
and see Rabkin, E. B.
Heinsius, Ernst. (357).
Hemphill, R. E. (1091).
Henderson, J. W., and Rucker, C. W. (348).
Henderson, M. D., see Mitchell, H. S.
Henderson, Thomas. (1463).
Henderson, Thomson. (595).
Herren, R. Y. (222).
Hertzau, Werner. (222), (1452).
Hessberg, R. (241).
Hesse, Erich. (826).
Hetherington, W., see Roepke, R. R.
Hilding, A. C. 703.
Hildreth, H. R. (600), **749.**
Hine, M. L. (1102).
Hirsch, Oscar. 949.
Hitz, J. B. (115), **1019.**
Hodes, R. (724).
Hoehne, K. (966).
Holden, David. 1314, 1315.
Holder, H. J., see McKelvie, A. R.
Holm, Ejler, and Lodberg, C. N. (717).
Holmes, W. J., and Hamman, G. C. (1102).
Holmgren, H., and Stenbeck, A. (835).
Holst, J. C. (237), (241), (1080).
Holstein, Theodore, see Meyer, S. J.
Holt, E. E., Jr., see Beach, S. J.
Holt, Helen, and Rötth, A. de. (350).
Hopkins, C. R. (1211).
Horay, Gusztáv. (969).
Hornbogen, D. P. **323.**
Horner, W. D. (1339).
Horrick, E. J. 695.
Hou, H. C. (1100).
Howard, P. J. (226).
Howard, W. H. (92).
Hruby, K. (1463).
Hudelo, André, (241).
Hudson, A. C. 570.
Hughes, W. L. 204.
Huntington, R. W., Jr., see Angevine, D. M.
Hurtault, J., Lando, M. X., and Garcío Querol, A. (112).
and Sverdlick, J. (232).
Hurwitz, Paul. 1314.
Hussey, R., see Roesler, H.
Hutchinson, Jonathan. (605).

Igersheimer, Joseph. 67, 72, (105), 1196, 1199.
Imre, Harry, (1095).
Imre, Josef. (839), (1077).
Imus, H. A., see Ogle, K. N.
Inciardi, J. A. (1077).
Irvine, M. B., see Irvine, Rodman.
Irvine, Rodman. (97), **779,** (1467).
and Irvine, M. B. (1461).
Isaacs, B. L., Jung, F. T., and Ivy, A. C. (242).
Ivy, A. C., see Isaacs, B. L.

Jackson, Edward. 74, 81, (116), **277,** 455, 579, (599), 705, 815, 1063, 1320, 1442.
Jackson, Sylvia, see Lyle, Keith.
Jacoby, Julius. (351).
Jacovlev, S. T. (222).
Jaeger, Antoine de. (1087).
Jaeger, Ernst. (727).
Jaensch, P. A. (224).
Jahnke, W. (715).
Jakovlev, P. A. (960).
James, R. R. (1343).
James, W. M. 939.
Jancke, G. (228), (1451).
Jasserson, F., and Morard, G. (720).
Jayme-Goyaz, G. G. **1281.**
Jeandelize, P., and Bardelli, P. (727).
Jeans, P. C., Blanchard, E. L., and Satterthwaite, F. E. (826).
Jendralski, Felix. (1449).
Jensen, A. F. **63.**
Jensen, C. D. F. (232).
Jensen, J. P. (109).
Jensen, V. A. (106).
Jervis, G. A. (102), (975).
Jess, Adolph. (1468).
Joannidès, T. (361).
Johnson, G. L. (827).
Johnson, L. V. **1232.**
and Eckhardt, R. E. (481).
and see Eckhardt, R. E.
Johnson, Robert. 447.
Jona, Sergio. (1228).
Jones, V. L. 206.
Joseph, E., see Kahn-Ostwald.
Judd, J. H. 331.
Judge, H. V. (837).
Julianelle, L. A. (90), 445.
and see Smith, J. E.
Jung, F. T., see Isaacs, B. L.
Jung, J. (228).
Juvanon, L., see Bailliart, P.

Kachan, Z. Z., see Katznelson, A. B.
Kahn-Ostwald, and Joseph, E. (1339).
Kalfa, C. A., and Brodskii, B. C. (1098).
Kalkutina, M. L. (1464), (1471).
Kalt, E. (350).

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Kamel, Sabri. (352), (1454).
Kaminskaja, Z. A. (596).
Kaminskaja-Pavlova, Z. H. (1464).
Kaminski, L. S. (1343).
Kantor, D. B. (1332).
Kanzer, M. G., see Bender, M. B.
Katznelson, A. B. (967).
  and Kachan, Z. Z., and Tufa, H. Z. (1075).
  and Strukov, A. I. (599).
Kaufman, S. I. (1080).
Kaye, Herbert. (1095).
Kayser, B. (1225).
Kazas, I. I. (1471).
Keiner, G. B. J. (106).
Keller, see Redslob.
Kelly, A. R., see Sydenstricher, V. P.
Kessler, Emma. (1085).
Kettler, J. R., and Rutherford, C. W. (90).
Keyser, G. W. (88), (108).
Khavin, A. O. (601).
Khayoutin, S. M. (592).
Khluser, G. P. (715).
Kholina, A., and Chernobilskaja, P. (1100).
Kilgore, G. L. (238), 1441.
Kindt, Peter. (98).
King, A., see Ford, F. R.
Kinmont, P. (960).
Kiparisov, H. M. (593), (720).
Kirby, D. B. (839), (1085).
  and see Rychener, R. O.
Kirk, R., see McKelvie, A. R.
Kisen, E. G. (465).
Kistner, F. B. (1330).
Klemens, F. (478).
  and see Reichling, W.
Klemme, R. M. **1000.**
Klien, B. A. **133.**
Klimovich, E. F., see Zimkin, H. B.
Kluever, H. C., and Coughlan, C. H. (715).
Knapp, Arnold. 1195, 1198, 1199.
Knapp, A. A. 214.
Knapp, F. N. (357).
Knapp, P., see Demole, V.
Knauber, Edna. **1022.**
Koke, M. P. (963).
Kolen, A. A. (219), (1340).
Kolenko, A. B. (219), (958).
Kopp, I. F. (228), (1225).
Kornzweig, A. L. 450.
Korobko, B. G. (972).
Korovitski. L. K., and Shein, I. S. (1330).
Kositzin, H. V. (1332).
Kosnun, V. I. (1096).
Koyanagi, Y. (967).
Kozshuknova, A. P., see Resnik, Z. I.
Kraehenbuehl, J. O. (1343).
Kramelashvili, H. G. (1331).
Krasilchikova, E. M. (975).
Krasnoretzki, P. A., and Lotin, A. V. (1226).
Krasnov, M. L. (1454), (1467).
Kravitz, Daniel, and Moehle, W. V. **527.**
Kronfeld, P. C. **51,** 694, 699, **1121.**
  and Lin. C. K., and Luo, T. H. **264,** (595).
Krotkov, H. V. (1461).
Krutova, A. H. (604).
Kuhn, H. S. (1102).
Kunitzkaja, L. V. (969).
Kurlat, Pedro. (976).
Kuvatov, G. G. (1459).
Kyrieleis, Werner. (242).

Lachman, R., see Muhlmann, V.
Lami, I., see Tiscornia, B. J.
Lancaster, W. B. **485, 619, 741,** (1073).
Landaburu, J. J., see Steinberg, I. R.
Landegger, G. P. (361).
Lando, M. X., see Hurtault, J.
Langer, Z. M. (968), (1077).
Larsson, Sven. (1340).
Lasky, M. A. (1448).
Lautsch, J., see Müller, H. V.
Laval, Joseph. (108), **408.**
Lavos, George. (606).
Law, B. B., and Philip, J. F. (1332).
Lazarev, E. G. (827).
Leathart, P. W. (969).
Lebensohn, J. E. 70, **1053,** (1091).
Leech, M. P., see Bloom W. A.
Lefkoeva, E. F. (1334).
Legras, A. M., and Wolters, D. H. F. (98).
Lehrfeld, Louis, (1093).
  and Brav, S. S. (968).
  and Breisacher, C. F. (465).
Leinfelder, P. J., and Black, N. M., Jr. **1115.**
Lemoine, A. N. (477).
Leonibus, Fernando de. (972).
Letchworth, T. W. (1226).
Levin, E., and Hanono, M. (1342).
Levin, O. L. 68.
Levina, L. S. (1465).
Levitt, J. M., and Blonstein, M. (104).
Levy, S., and Little, O. A. G. (827).
Lewis, J. T. (1345).
Lewis, Park. (116).
Lieto Vollaro, A. de. (108).
Lightbody, H. D., see Calvery, H. O.
Lijo Pavia, J. (88), (102), (216), (471), (471), (474), (827), (827), (828), (828), (957), (1090), (1090), (1098), (1098), (1229).
  and Cerboni, F. C. (462), (473).
  and Tartari, R. A., and Cerboni, F. C. (828).
Lillie, W. I. (476).
Lin, C. K., see Kronfeld, P. C.
Lindeman, Olaf. (357).
Lindner, K. (357).
Lisch, Karl. (1472).
Little, O. A. G., see Levy, S.
Lloyd, R. I. (607).
Lodberg, C. N., see Holm, Ejler.
Loens, M., see Bartels, M.
Loewenstein, Arnold. (93), (361), (968).
  and Steel, Janet. **731.**
Lokshin, S. I. (602), (837).
Long, J. C., see Danielson, R. W.
Long, P. H., see Haviland, J. W.
Longhena, Luisa. (1212).
Lopes, Octacilio. 85.
Lordan, I. P. (1453).
Lowry, J. T. (1462).
Lotin, A. V., see Krasnoretzki, P. A.
Lucké, B., and Schlumberger, H. (109), (110).
Luckiesh, Matthew, and Moss, F. K. **423, 1061,** (1217).

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Ludvigh, Elek. **303,** (1217).
Luedde, W. H. **40,** (971).
Lugossy, Gyula. (1454).
Lugossy, Julius. (226), (467), (722).
Lukić, Djordje, J. (1454).
Luo, T. H., and Chang, E. (1075).
and P'an, S. Y. (232).
and see Kronfeld, P. C.
Luschinskii, G. F., and Gilbert, G. P. (958).
Lushin, M. I., see Zimkin, H. B.
Lutman, F. C. 559, 559.
Luzsa, Andreas. (478), (1081).
Luzsa, Endre. (970).
Lyle, T. K., and Jackson, Sylvia. 588.
and McLean, D. W. (1077).
Lyman, R. S. (463).
Lythgoe, R. J., see Broda, E. E.

McCall, E. F., see Cosgrove, K. W.
McCurdy, M. W., see Burleson, J. H.
MacDonald, A. E. (93), (598).
McDonald, Robb, and Adler, F. H. (243).
and see Adler, F. H.
McFadyen, John. (358).
McGuire, H. H., and McGuire, W. P. **657,** (970).
McGuire, W. P., see McGuire, H. H.
Machado, N. R. (1221), (1221).
Machlin, I. M. (1098), (1340), (1344).
McKee, S. H. (840), (1100).
McKelvie, A. R., Kirk, R., and Holder, H. J. **1035.**
McKeown, H. S. (243), (598), (1469).
McKinney, I. M., and Frocht, M. (233).
McKinney, J. W. (968).
McLean, D. W., see Lyle, T. K.
McLean, J. M. **46.**
MacMillan, J. A. (1093).
McNair, G. S. (1472).
and see Bettman, J. W.
Macnie, J. P. (721), (835).
MacPherson, W. A., see Gifford, S. R.
Madigan, L. F., see Ogle, K. N.
Maddren, R. J. **1307.**
Maggi Zavalia, Juan. (1102).
Magitôt, A. (219).
and Dubois-Poulsen, A. (98), (216), (725).
Maisel, I. D. (465).
Malbran, Jorge. (961).
Malling, H. (93).
Malone, J. Y. (88).
Mandelbaum, Joseph, and Mintz, E. U. **1241.**
Mann, Ida. and Pullinger, P. D. (113).
Marback, Hector. (471)
Marchesani, O. (1342).
Marioth. (358).
Marmorstein, F. F., see Dashevskii, A. I.
Márquez, M. (717).
Marshall, W. H., see Talbot, S. A.
Martin, G. H. **287.**
Martin, J., see Davis, L.
Martin, J. P., and Savin, L. H. (1335).
Martin, L., and Pennell, V. (961).
Martinez Hinojosa, Francisco. (1345).
Marx, H., see Reichling, W.
Marx, P., see Redslob, E.
Mastier, P., and Farnarier, G. (349).
Mastrangelo, Giuseppe. (463).
Mattos, W. B. (1218), (1228).
Maude, J. D. (222).
Maxwell, J. P. 1318.
Maxwell, J. S. **298.**
and see Traugh, G. H.
Maxwell, J. T. (1453).
May, C. H., and Perera, C. A. **1071.**
Meding, C. B. (238).
Medvedjev, H. I. (1340).
and Zatz, L. B., and Zolotareva, M. M. (602).
and Zolotareva, M. M. (1098).
and see Paneva, V. A.
Meek, R. E. 204, (352).
Meeker, Louise. 1314.
Meesmann, A. (463).
and Bachmann, W. (229).
Meinberg, W. H. 1438.
Meisner, T. (358).
Meister, E. J. 74.
Meksina, F. M. (600).
Mengel, W. G. 562.
Menshutin, M. A . (1222).
Merkulov, I. O., and Zabotinskaja, P. H. (601).
Mescherski, H. H. (596).
Métivier, V. M. **1029, 1265.**
Metzger, Ernst. 1316.
Meves, Harald. (1222), (1469).
Mexina, F. M. (1087).
Meyer, F. W. (234), (1342).
Meyer, G. P., and Reber, Jacob. 335.
Meyer, I. C. (1229), (1344).
Meyer, Karl, Block, H. S., and Chamberlain, W. P., Jr. **60.**
and Chaffee, Eleanor. (93).
Meyer, S. J., and Holstein, Theodore, **247,** 944.
Michaelson, I. C., and Campbell, A. C. P. (1088).
Michailov, A. X. (837), (837).
Mielke, Sophus. (358).
Mihályhegyi, Géza. (717), (1459).
Mikaelian, P. X., Zyck, H. F., and Vigodskaja, I. G. (233).
Mikhailov, M. H. (361).
Miklos, A. (1214), (1225).
Milaras, T. (828).
and see Redslob, E.
Milner, J. G. (973).
Mineev, P. A. (842).
Minsky, Henry. 1440.
Mintz, E. U., see Mandelbaum, Joseph.
Mineev, T. (590).
Mion, R. H., see Baudouin, A.
Missiroli, G. (715).
Mitchell, H. S., Cook, G. M., and Henderson, M. D. (473).
Mitzkevich, L. D. (593), (1212), (1214).
Moehle, W. V., see Kravitz, Daniel.
Moers-Messmer, Harold. (718).
Moffatt, P. M. 807, 808.
Moncreiff, W. F., and Scheribel, K. J. **282,** 1315.
Moorhouse, J. H. (1330).
Morard, G , see Jasserson, F.
Moret, R. L., see Tiscornia, A.
Morgan, A. L. (1333).

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Morgan, O. G. (463).
Morrison, W. H. (1469).
Moss, F. K., see Luckiesh, M.
Mould, W. L. (468).
Moura Compos, de. (244).
Mueller, E. F., see Thorson, J. A.
Mügge, Felix. (463).
Muhlmann, V., Ré, B. V., and Lachmann, R. (100).
Muir, E. B. **879.**
Mullen, C. R. (1076).
and Weintrob, J. R. 1434.
Müller, H. V., Schultz, H. J., and Lautsch, J. (244).
Mulock Houwer, A. M. (606).
Muncy, W. M. (219).
Muradian, E. A. (590).
Murphy, E. S. (102).
Musabeili, Y. (1103).
Muskhelishvili, S. V. (1088).
Myerson, A., and Thau, W. (235).
and see Thau, W.

Nano, H. M., see Tiscornia, A.
Nastri, Francesco. (238).
and Basile, G. (110).
Nausch, Bernhard. (722).
Nazarov, V. V. (358).
Neher, E. M. (1455).
Németh, Lajos. (459).
Nicholls, J. V. V. (1344).
and see Tooke, F. T.
Nicolacopoulos, J. (352).
Nikolaeva, M. C. (1093).
Nilson, E. L., set Berens, Conrad.
Nižetić, Z. (352), (837).
Nordenson. 452.
Nordmann, Jean. (840).
Nowak, E. (718).

O'Brien, C. S. 1444.
and Allen. J. H. (244).
Ochapovskaja. H. V. (1455).
Ochi, S. (1455).
O'Connor, Roderic. (93), **640,** (960).
O'Day, Kevin. (217).
Offret, G., see Desvignes, P.
and see Velter, E.
Ogle, K. N., Imus, H. A., Madigan, L. F., Bannon, R. E., and Wilson, E. C. (461).
Ohm, J. (235), (719).
Oishi, S. (721).
Oláh, Emil. (226), (227).
Oliva, R. O., see Zavalia, U. A.
Ornsteen, A. M., see Scheie, H. G.
O'Rourke, D. H. 702.
Osburn, John. 1441.
Ostmann. (100).

Pacalin, G. (229).
Pacheco Luna, R. (481).
Paez Allende, Francisco. (474), (1218).
Pahwa, T. R. (1096).
Palin, Anthony. 806.
Paltzeva, Z. A. (1096).
P'an, S. Y. (959).
and see Luo, T. H.

Paneva, V. A., and Medvedjev, H. I. (1453).
Panneton, Philippe. **314.**
Papolczy, Ferenc. (840).
Parker, W. R. (233).
Parkhill, E. M., and Benedict, W. L. **1354.**
Parlange, see Talbot.
Pascal, J. I. (223), (223), 441, 822, (1218), **1308.**
Pascheff, E. (968).
Pasternak, H. G. (1331).
Patch, E. M. (1085).
Patek, A. J., Jr., and Haig, C. (976).
Paufique, Louis. (347).
and see Bonnet, Paul.
Paula Santos, B. (1328).
Pavišić, Zvonimir. (479).
Payne, B. F. **395.**
Pennell, V., see Martin, L.
Pereira, R. F. (99).
Pereira Gomes, J. **1144.**
Perera, C. A. (94), (113), (843).
and see May, C. H.
Pergola, Alfredo. (1340), (1344).
Peter, L. C. 712.
Peters, E., see Hansen, K.
Petronio, G. (1228).
Petrosiantz, E. A. (468), (593).
Petrov, D. G. (459).
Petrov, M. D. (459).
Petrunia, S. P. (594).
Pfingst, A. O. **257,** (590).
Philip, J. F., see Law, B. B.
Piatt, A. D., see Posner, I.
Pick. (590).
Pieck, C. M. E. (110).
Pillat, A. (465), (604), (1078).
Pillet, P., see Borsotti, I.
Pincus, M. H. **503.**
Pinho, Edson. (1214).
Platova, Z. I. (1467).
Plitas, P. S. (1340).
Pokhisov. H. I. (353), (600).
Pokrovskii, A. I. (235), (1335), (1335), (1336).
Popov, M. Z. (1465).
Poschin, B. P. (229).
Posner, Adolph. 569.
Posner, I., and Piatt, A. D. (350).
Post, L. T. 445, 582, 708.
Post, M. H. 206, **624,** (959).
Posthumus, R. G. (113).
Poštić, Svetozar. (1098), (1459).
Prado, Durval. (1218), (1344).
Prangen, A. D. **413.**
Preobraszenski, B. B. (235), (716).
Preston, F. E. (1451).
Pritikin, R. I. 1311, 1311, 1311.
Proksch, Marie. (475).
Puchkovskaja, H. (592).
Puech, P., Bonnet, R., and Guillaumat, L. (347).
Puglisi-Durante, Giovanni, and Rebaudi, F. (359), (466).
Puig Solanes, Magin. (1222).
Pullinger, P. D., see Mann, Ida.

Que, K. L. (90).
Quevedo, Arturo. **1185.**

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Quilliam, J. P., see Davson, H.
and see Duke-Elder, Sir W. S.

Raab, C. M. (220).
Rabkin, E. B., and Heglo, L. G. (1218).
Radnót, Magda. (1093), (1099).
Radzichovskii, B. L. (468), (591), (1076), (1226).
Rambo, V. C. (90).
Rameev, P. C. (359).
Ramsay, A. M. (471).
Rand, Gertrude, see Ferree, C. E.
Raski, K. (721).
Rasmussen, A. T. 1207.
Ratinova, K. A. (973).
Rauh, Walter. (1451).
Ré, B. V., see Muhlmann, V.
Rea, R. L. 821, (1088).
Read, W. T., Jr., see Barnshaw, H. D.
Rebaudi, F., see Puglisi-Duranti, G.
Reber, Jacob. **161.**
and see Meyer, G. P.
Reca, A. B. (1223).
Redslob, E., and Keller, (719).
and Marx, P., and Dieffenbach, P., and Milaras, T. (721), (833).
Reese, A. B. (104), **386, 497,** (606), (1082).
and Yaskin, J. C. **544,** (961).
Regoli, Attilio. (1333).
Reichling, Walther. (723).
and Klemens, F. (1345).
and Marx, H. (1331).
Reinhorn, John. 570.
Reinus, A. M., and Sharkovski, I. A. (1092).
Reis, Carolina. (1455).
Reiser, K. A. (1079), (1459).
Reiss, P. (847).
Renner, see Harms, H.
Resnik, Z. I., and Kozshuknova, A. P. (1333).
Rethmeier, B. J., see Boshoff, P. H.
Riddell, W. J. B. (597), (847), (1328).
Rieken, Hans. (474).
Riser, R. O. **874.**
Rittenhouse, E. A. (461).
Riwchun, M. H. **798.**
and DeCoursey, E. (1096).
Roaf, H. E., see Hay, J. C.
Robbins, A. R. **556,** (1093).
Rocha, Hilton. (1344).
Rochat, G. F. (105).
Rochat, G. F., Jr. (98).
Rodigina, A. M. (1345).
Roelofs, C., see Faber, E.
Roesler, H., Gibson, G., and Hussey, R. (102).
Roepke, R. R., and Hetherington, W. A. (233).
Roetth, Andrew de, see Rötth, András de.
Roggenkämper. (230).
Rokitzkaja, L. V. (971).
Rolett, D. M. (479), 566.
Rollenberg Sampaio, J. M. (1223).
Rollin, A. (244).
Rones, Benjamin. (94), (1459).
and see Calvery, H. O.
Roper, K. L. (962).
Ros, Antonio. 1326.
Rosenbaum, H. D., see Wiener, Meyer.
Rosenbaum, J. (1094).
Rosenberg, L., and Rosenberg, J. (90).
Rosenblum, M. E. (1099).
and see Bogoslovski, A. I.
Rosenthal, B. C. 205.
Roslavtzev, A. V. (1219).
Rössler, Fritz, (718).
Rothbard, Sidney, see Berens, Conrad.
Rötth, András de. **20,** (94), 698.
and see Holt, Helen.
Rousseau, and Thomas. (832).
Roux, J., see Dejean, C.
Rubbrecht, R. (91), (723).
Rubino, Agostino (1462).
and Simonelli, M. (1223).
Rucker, C. W., see Henderson, J. W.
Ruedemann, A. D., and Zeiter, W. J. (220).
Rugg-Gunn, A. (1212).
Rumantzeva, A. F. (1331).
Rumbaur, W. (973), (1336).
Rutherford, C. W. 713.
and see Kettler, J. R.
Rychener, R. O. **322.**
and Kirby, D. B. (94).

Saba, Gaetano. (1219).
Saburov, G. I. (359).
Sachs, Erich, and Heath, Parker. **34.**
Sag, H. O. (1089).
Salit, P. W. **191, 310,** (727), (973).
and see Swan, K. W.
Sallmann, Ludwig von. (829), (840), 1317, **1349.**
and see Friedman, Benjamin.
Samoilov, A. I. (1470).
and Federov, H. T., and Davidov, V. G. (1089).
Samokhina, E. F. (1081).
Samuels, Bernard. (1090).
Sanchez Bulnes, Luis. (1227).
Santa Maria, J., see Vila Ortiz, J. M.
Saradarian, A. V. (479).
Sarrouy, see Schousboë, F.
Satanowsky, Paulina. (1462).
Satterfield, G. H., see Caveness, H. L.
Satterthwaite, F. E., see Jeans, P. C.
Sattler, C. H. (464).
Sauberman, G. B. C., and Schmidt, A. E. (1214), (1214).
Savin, L. H. (1223).
and Tyrell, T. M. (459).
and see East, T.
and see Martin, J. P.
Scardaccione, Mario. (99), (105), (1219).
Schaub, C. F. 945, 1312, 1312, 1313.
Scheerer, P. (230).
Scheie, H. G. (98).
and Ornsteen, A. M. 561.
and Souders, B. F. (1449).
and see Adler, F. H.
Scheribel, K. J., see Moncreiff, W. F.
Schiff-Wertheimer, S., and Galan, R. (244).
and see Bailliart, P.
Schlaeppi, V. (972).
Schley, H. (359).
Schlivek, K., see Goldberg, H. K.
Schlumberger, H., see Lucké, B.

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Schmelzer, Hans. (235), (1082).
Schmid, A. E., see Sauberman, G. B. C.
Schmidt, Martin. (968).
Schousboë, F., and Sarrouy. (349).
Schoute, G. J. (106).
Schreuder, M. (95).
Schubert, F., see Hartinger, H.
Schujman, S., see Decoud, A. C.
Schultz, H. J., see Müller, H. V.
Schultz, V. A., see Bursuk, G. G.
Schwartz, F. O. 941.
Schwartz, M., see Allen, F.
Sédan, Jean. (833), (843).
Seif, H. A. (1467).
Seidel, E. (103), (116).
Seidenari, Renato. (350), (458), (1082), (1214).
Semeraro, E. (1221).
Sergievskii, L. I., and Bilit, M. B. (962).
Shafar, J., see Worster-Drought, C.
Shandeling, P. D. 558.
Sharkovskii, I. A. (599).
and see Peinus, A. M.
Shartz, S. E. (597), (1079).
Shaw, W. J., see Bloom, W. A.
Sheftel, A. G. (976).
Shein, I. S. (591), (596).
and see Korovitski, L. K.
Sheplakova, V. M. (1336).
Sherwood, Robert, see Bane, William.
Shevchuk, I. J. (596).
Shively, F. L., see Gardner, W. J.
Shone, S. (347).
Sie-Boen-Lian, B. L. (91), (1096).
Sikharoulidze, I. A. (1092).
Silva Costa, Alvaro da. (838).
Silva, Linneu. (1221).
Simeone, F. A. 200.
Simonelli, M., see Rubino, A.
Simpson, G. V., see Verhoeff, F. H.
Simpson, R. K. (1219).
Singer, E., see Evans, J. N.
Siotto, Giovanni. (1336).
Sironi, Luciano. (110).
and see Bossalino, G.
Sjögren, Henrik. (838).
Skorodinskaja, V. V. (1225).
Skvirskaja, Z. M., see Gekker, I. P.
Sloane, A. E. (606).
Smart, F. P. (461).
Smelanski, P. I. (1336).
Smelanskii, I. A. (600).
Smelser, G. K. **680.**
Smith, Byron. 450, 451.
Smith, Carroll. (354).
Smith, H. C. (601).
Smith, J. E., Julianelle, L. A., and Gamet, J. H. **174.**
Smith, J. W. **537.**
Snell, A. C. 211.
Sniderman, H. R. (477), **675.**
Sobacke, Lucille, see Berens, Conrad.
Solares, Amiceto. **900.**
Sondermann. (472).
Sorsby, Arnold. (103), (598), (602).
Souders, B. F. 75, (355).
and see Scheie, H. G.
Sourdille, G. P., and David, M. (347).
and Duval, E., and Gendron. (238).
Southerland-Campbell, H. (348).
Spaeth, E. B. **186,** 203, 954, (1099), 1437.
Spaski, V. I. (466).
Spector, C. A. (230).
Spivakovskii, D. H. (1340).
Spratt, C. N. (479).
Stallard, H. B. (108), (108), (355).
Stanford, C. E. (847).
Stark, E. K. **1299.**
Statti, L. W. (1215).
Staz, L. (841).
Steel, Janet, see Loewenstein, Arnold.
Steele, E. J. P. (344).
Steinberg, I. R., Charosky, L., and Landaburu, J. J. (466).
Stenbeck, A. (106).
and see Holmgren, H.
Stenstrom, W. K., Burch, E. P., and Walsh, F. M. (959).
Sternes. 943, 943, 944, 944.
Stewart, C. P. (1470).
Stewart, Marshall. (235).
Stockwell, Eunice. 946.
Stokes, W. H. (472).
Stone, J. B., and Courtney, R. H. (1225).
Stone, L. S., and Dinnean, F. L. (238).
and Ellison, F. S. (362).
Strachov, V. P. (1337).
Stromberg, Günter. (344).
Strukov, A. I., see Katznelson, A. B.
Suárez Gabard, Santiago. (1455), (1462).
Suden, C. T., see Tum Suden, C.
Suganuma, Sadao. (968).
Sugar, H. S. **851.** (1083).
Sukhov, S. V. (974).
Sullivan, G. L. **936.**
Sverdlick, Jose. 821, (1221), (1453).
and see Hurtault, J. B.
Sverdlin, V. I. (591).
Sverdlow, D. G. (716).
Sviadocz, B. I., see Baltin, M. M.
Swab, C. M. (597).
Swan, K. C., and Salit, P. W. **611.**
Swigert, J. L. 74.
and Weston, Horace. 330.
Sydenstricker, V. P., Kelly, A. R., and Weaver, J. W. (1079).
Sydokevich, D. I. (361).
Svsi, R. (1460).
Szinegh, Béla. (724).

Talbot, Parlange, and Duroux. (1341).
Talbot, S. A., and Marshall, W. H. **1255.**
Talkovskii, S. I. (1346).
Tartari, R. A., see Lijo Pavia, J.
Tatár, József. (106), (480), (834), (959), (1455).
Taylor, E. M. (99).
Telesheva, A. I. (724).
Terrien, F., Azerad, E., and Voisin, J. (348).
Terry, T. L. (111).
Tertsch, R. (1225).
Tettamanti, Julio. (230).
Thau, W., and Myerson, A. (236).
and see Myerson, A.
Theobald, G. D. (597), **1132.**
Theodore, Frederick. 1438.

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Thier, P. F. X. (106).
Thomas, Maxwell. (1460).
Thomas, and Weill. (1226).
and see Rousseau.
Thomas, J. W. T. (95).
Thometz, H. M. (100).
Thompson, Richard. (1094).
and Gallardo, Edward. **635.**
Thordarson, U. (725).
Thorner, M. W. 75.
Thorson, J. A., and Mueller, E. F. (836).
Thygeson, Phillips. (964).
Tichova, V. A. (466), (1099).
Tichomirov, P. E. (1466).
Tikhomirov, A. A. (976).
Tillé, H., see Bailliart, P.
Tiscornia, A., Moret, R. L., and Tiscornia, B. J. (964).
and Nano, H. M. (478).
Tiscornia, B. J. and Lami, I. (1337).
and see Tiscornia, A.
Tita, Carlo. (724).
Todoroff, D. A. (724).
Tokareva, B. A. (602).
Tooke, F. T., and Nicholls, J. V. V. (829).
Tooker, C. W. 443.
Török, Ervin. 1197, 1198.
Torres Estrada, Antonio. (91), (468), (477), (480), (1222), (1227), (1329).
Town, A. E. 443.
Townes, C. D. 955.
Trantar, N. G. (1085).
Trantas, A., and Trantas, Nico. (343), (468).
Trantas, Nico, see Trantas, A.
Traquair, H. M. (480), (1094).
Traugh, G. H., and Maxwell, J. S. **800.**
Troncoso, M. U. 83, **1396.**
Tsopelas, B. (355).
Tufa, H. Z., see Katznelson, A. B.
Tumantzeva, H. F. (343).
Tum Suden, C., (974).
and Wyman, L. C. (239).
Turner, J. W. A. (1090).
Tüshaus, Rainer. (355).
Tyrrell, T. M., see Savin, L. H.
Tzikulenko, K. I. (1345).

Urbanek, J. (1467).
Uribe Troncoso, M., see Troncoso, M. U.
Urrets Zavalia, see Zavalia, A. U.

Vail, Derrick. 79. (95). 210. 339, **403.** 456, 585, 711. 819, **920,** 951. 1069, 1206, 1325, 1445.
Valerio, Mario. (724).
Valle, Sergio. (1224).
Van Bergen, T. M. 558, 559.
Vancea, P. (1103).
Van der Hoeve. J. (105).
Van Lint, A. (99).
Varshavskaja, P. P. (460).
Vasquez Barrière, A. (974).
Veasey, C. A., Jr. (605), (1101), (1215).
Veil, Prosper. (841).
Velhagen, K. T. (233), (344).
Velhagen, K., Jr. (474).
Velter, C. L. (594).
Velter, E., and Offret, G. (832).
Veltitzev, F. (1091).
Vengrshenovski, T. C. (353).
Verhage, J. W. C. (962).
Verhoeff, F. H. (458), (964), (1074).
and Simpson, G. V. (345).
Verrier, L. (362).
Viallefont, H. (223).
and see Dejean, C.
Victor, E., see Broda, E. E.
Vigodskaja, I. G., see Mikhaelian, I. G.
Vila Ortiz, J. M., and Santa Maria, J. (600).
Vilovskaja, K. E., see Askalonova, T. A.
Viramontes, Luis. (475).
Vishnevskii, H. A. (1212).
Viswalingam, A. (1226).
Vladichenskii, A. (959).
Vogt, Alfred. (829).
Voisin, J., see Terrien, F.
Volokitenko, A., see Filatov, V. P.
Vorisek, E. A. **928, 1014.**
Vrolijk, M. (1460).

Waardenburg, P. J. (107).
Waddy, G., see Brodsky, I.
Wagener, H. P. (103), 325, (345).
and see Watkins, C. H.
Walker, A. E., see Halstead, W. C.
Walker, Charles, Jr. 330.
and Weston, Horace. 701.
Walker, J. P. S. (1460).
Walsh, F. B., and Ford, F. R. (217).
Walsh, F. M., see Stenstrom, W. K.
and see Ford, F. R.
Wasserman, I. A. (230), (595), (1080).
Watkins, C. H., Wagener, H. P., and Brown, R. W. **1374.**
Weaver, J. W., see Sydenstricker, V. P.
Weber, Ernst. (223), (1219).
Webster, J. E., see Weinberger, L. M.
Weekers, Roger. (231), (836), (974).
Weem. 1313.
Weill, see Thomas.
Weill, G. (843).
Weinberger, L. M. 947.
and Adler, F. H., and Grant, F. C. (476).
and Webster, J. E. (834).
Wells, D. M. (227).
Wendling, Meinhard. (345).
Werdenberg, E. (830).
Werner, Sigurd. (725).
Weskamp, Carlos. (591), (1345).
Weston, Horace, see Bane, William.
and see Swigert, J. L.
and see Walker, Charles, Jr.
Wetzel, J. O. **164.**
Whalman, H. F. (91).
Wheeler, J. R. (957).
White, G., and Hassard, C. E. A. (1470).
White, J. W. **156,** (464), 812.
Whiting, Maurice. (930).
Whitney, E. L., see Brown, W. M.
Wiegersma, G. (236).
Wiener, Meyer. 1439, (1468).
and Rosenbaum, H. D. **1384.**
Weintrob, J. R., see Mullen, C. R.
Wies, F. A. **439.**
Williams, J. F. 1326.
Wilson, Clinton. (959).
Wilson, E. C., see Ogle, K. N.

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Wilson, R. P. (467), (1456), (1456), (1456), (1460), (1460), (1460), (1468), (1472).
Winkler, Adolf. (830).
Wolf, E., see Crozier, W. J.
Wolfe, Otis, and Wolfe, R. M. (1468).
Wolfe, R. M., see Wolfe, Otis.
Wolff, Eugene. 587, (1103), (1103).
Wolters, D. H. F., see Legras, A. M.
Wong, R. T. (830), (1461).
Woodruff, F. E. 84.
Woods, A. C. 1067.
and Burky, E. L. (845).
and see Guyton, J. S.
Worster-Drought, C., and Shafar, J. (348).
Wright, R. E. (467).
Wÿers, H. J. G., see Goedbloed, J.
Wyman, L. C., see Tum Suden, C.

Yanes, T. R. (343).
Yanez, E. G. (602).
Yarbrough, M. E., see Dann, W. J.
Yaskin, J. C., see Reese, W. S.
Yazujian, D. M. (480).
Youfa, H. Z. (1450).
Young, J. H. (350).
Yousefova, F. I. (1342).
Youshkin, Y. I. (607).
Yudkin, Simon. (1089).

Zabotinskaja, P. H., see Merkulov, I. O
Zakharof, A. P. (591).
Zakrzevskaja, E. I. (603).
Zamberg, D. A. (843).
Zamkovskii, I. G. (1450).
Zarembo, A. (343).
Zatz, L. B., see Medvedjev, H. I.
Zavalia, A. U., and Anquin, M. H. de. (844).
and Oliva, R. O. (974).
Zeiter, W. J., see Ruedemann, A. D.
Zenkina, L. V. (975).
Zimin, V. I. (1083).
Zimkin, H. B., Klimovich, E. F., and Lushin, M. I. (960).
Ziporkes, Joseph. (345).
Zolotareva, M. M., see Medvedjev, H. I.
Zorab, E. C. (1089).
Zshiv, V. V. (964).
Zur Nedden, M. (469).
Zyck, H. F., see Mikhaelian, P. X.

SUBJECT INDEX

Compiled by EMMA S. BUSS

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Abducens, insufficiency, congenital, 694.
paresis of, (358), (833).
surgical treatment of, 565.
Abscess, episcleral, (1460).
Abstracts—
1. General methods of diagnosis, (215), (458), (714), (957), (1211), (1447).
2. Therapeutics and operations, (217), (459), (714), (957), (1212), (1448).
3. Physiologic optics, refraction, and color vision, (88), (220), (460), (716), (959), (1215), (1450).
4. Ocular movements, (89), (223), (462), (719), (961), (1073), (1219), (1451).
5. Conjunctiva, (89), (224), (464), (590), (720), (962), (1074), (1222), (1454).
6. Cornea and sclera, (91), (227), (467), (592), (722), (965), (1076), (1224), (1456).
7. Uveal tract, sympathetic disease, and aqueous humor, (95), (231), (469), (595), (724), (969), (1080), (1226), (1461).
8. Glaucoma and ocular tension, (99), (234), (472), (596), (725), (970), (1081), (1226), (1462).
9. Crystalline lens, (100), (236), (343), (473), (596), (726), (972), (1083), (1227), (1466).
10. Retina and vitreous, (101), (239), (344), (474), (597), (824), (975), (1085), (1228), (1328), (1468).
11. Optic nerve and toxic amblyopias, (104), (346), (475), (598), (830), (976), (1090), (1329), (1470).
12. Visual tracts and centers, (105), (348), (475), (599), (832), (1091), (1471).
13. Eyeball and orbit, (106), (349), (476), (599), (834), (1092), (1330), (1472).
14. Eyelids and lacrimal apparatus, (107), (351), (477), (600), (836), (1093), (1332).
15. Tumors, (108), (353), (478), (600), (838), (1094), (1333).
16. Injuries, (111), (355), (479), (601), (841), (1097), (1337).
17. Systemic diseases and parasites, (113), (359), (481), (603), (844), (1099), (1341).
18. Hygiene, sociology, education, and history, (114), (361), (605), (846), (1101), (1343).
19. Anatomy, embryology, and comparative ophthalmology, (116), (361), (607), (847), (1103), (1345).
Accessory nasal sinuses, see Nasal accessory sinuses.
Accidents, retinal detachment and, (240).
and see Injuries.
Accommodation, astigmatic, (1216).
and chromatic near test, (718).
effect of Meckel's ganglion on, (971).
at eighty years of age, 74.
Accommodation, (*continued*).
functions of relative, **423.**
Helmholtz theory of, objections to, 452.
measurement of, (960).
pocket rule for, (959).
mechanism of, in subluxated lenses, **40.**
reflex, visuopsychic apparatus and, (222).
significance of, in refraction, 560.
spasm of, (1215).
Acetylcholine, content of retina, (1086).
effect of, on embolism of retinal blood vessels, (975).
Achromatopsia, see Color blindness.
Acrichinin therapy, effect of, on eyeball, (1213).
Actinomyces of orbit, (836).
Actinomycosis, facial and orbital, (350).
Adair-Dighton's syndrome, see Syndrome.
Adaptation, dark, effect of thyroid extract on, (976).
hemeralopia, (242).
measurement of, (825).
nutritional factors in, (1470).
in optic-nerve disease, (1471).
after surgery in glaucoma, (971).
sensitivities of the color receptors measured by, **1241.**
tests, clinical evaluation of, 77, (243), (714).
new, (1089).
standardization of, (244).
and vitamin A, (344), 703, (826), (976), (1086), (1470).
light, in vitamin-A deficiency, (344).
Adaptometer, (975).
readings in avitaminosis, (1086).
Adenoma, hypophyseal, (1332).
and syndrome of cavernous sinus, (476).
sebaceum, phakoma retinae and, (478).
Adenocarcinoma of lacrimal gland, (600).
Adie's syndrome, see Syndrome.
A-dinitrophenol, effect of, on dark adaptation, (976).
Adrenalin, desensitization with, (219).
effect of, on Rayleigh's interval, (222).
lens opacities induced by, (239), (974).
Albino, senile cataract in, **798.**
Albucid, see Sulfanilamide.
Alcohol, effect of, on vision, (599).
Allergy in ophthalmology, (360), **759,** (1469).
cataract with eczema, (973).
conjunctival, (1074).
corneal, (967), (1078).
eyelids and conjunctiva as shock tissues in, (1093).
interstitial keratitis, syphilitic, and, (966).
tuberculin, (1454).
Alopecia in traumatic iridocyclitis, (601).
Amaurosis, puerperal, (1471).
Amaurotic idiocy, family, (102), (975), (1228).
juvenile, disturbances of water-salt metabolism in, (597).
Spielmeyer-Vogt, (827).

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Amblyopia, toxic, 331.
quinine, (346).
sodium diphenyl hydantoinate (Dilantin), (104).
tobacco, and myopic macular changes, (475).
wood-alcohol, blood transfusions, etc., in recovery from, (1470).
and see Abstracts, section eleven.
Amblystoma, punctatum, exchange of eyes of, with Triturus viridescens, (362).
tigrinum, cataracts produced in, (1085).
American Academy of Ophthalmology and Otolaryngology. Section on Ophthalmology, 1941 meeting, 1445.
American Board of Ophthalmology, model case histories, **690, 936.**
American Medical Association, Section on Ophthalmology, 1941 meeting, 820.
American Ophthalmological Society, 1941 meeting, 819.
1940 transactions, 586.
Anatomy, embryology, and comparative ophthalmology, (116), (361), (607), (847), (1103), (1345).
Anemia, experimental, ocular changes in, (1337)
pernicious, optic atrophy with, (1090).
retrobulbar neuritis, an early sign of, (347).
Anesthesia, evipal-sodium, (459).
pentothal sodium, (1215).
retrobulbar, proctocaine, for intractable pain, (459).
spinal, paralysis of external rectus muscle after, (462), (719).
Aneurysm, arteriovenous, **800.**
carotid-cavernous, pulsating exophthalmos, **365.**
cirsoid, of orbit, (477).
of internal carotid artery, with calcification, (105).
of small retinal arteries, with Leber's disease, (344).
Angioendothelioma of disc, (110).
Angioid streaks of retina, see Retina.
Angioma of orbit, (1335).
Angiomatosis retinae, (240), (841), (1336).
treatment, (1095).
Angioscotoma, changes in, due to Brucellosis, 342.
Angioscotometry, in choked-disc diagnosis, (216).
diagnostic significance of, (1212).
Angiospasm of visual cortex, (160), (834).
Aniseikonia, (222), 1321.
clinics, 708.
Anisochromatopsia, congenital, (88).
Anisometropia, prism compensation in, (1216).
Anisotropism in elasticity of corneal tissue, (95).
Anomalies of eye, (223).
binocular, (1081).
of lacrimal canaliculus, (108).
in congenital and acquired blindness, (350).
of lid, 698.
Anomaloscope, Nagel, (222).
Anoxia in scleritis and episcleritis, (965).
Anterior chamber, agglutinins in, after irritation of peripheral nerves, (233).
cyst in, free, 329.
difference of tension in, and in posterior chamber, (1081).
foreign body in, delayed removal of, (356).
nematode worm, (113).
implantation of hormonal substances in, (470).
limits of tyndallimetry, in, **51,** 699.
pH in diffusion of alkaloids in, (969).
Aphakia, retinal detachment in, (237).
visual threshold for blue in, (716).
Aqueous humor, appearance of sulfanilamide, in (232).
cyst in, free, 329.
glucose in, (836).
and ocular inflammations, (1461).
osmotic relation between, and blood plasma, (233).
protein content of, **1121.**
of re-formed, **264,** (595).
regeneration of, (469).
secretion of, in mammalia, (595).
sulfanilamide appearance in, (1449).
sulfapyridine distribution in, **60.**
vitamin-C content, (1466).
and see Abstracts, section seven.
Arachnodactyly, histologic examination of eye in, (597), **1132.**
lens ectopia in, see Marfan's syndrome.
Arachnoiditis, chronic local, prechiasmal syndrone with, (476).
optic chiasm, cystic, (832).
diagnosis and treatment, (832).
syphilitic, 945, 1313.
optic atrophy due to, treatment, **119.**
Argyll Robertson pupils, see Pupil.
Ariboflavinosis, ocular manifestations of, (481), (1079).
Arteriosclerosis, clinical significance of retinal and choroidal, 325.
and diabetic fundus, (1100).
Arthritis, juvenile atrophic with corneal involvement, (1461).
poly-, rheumatoid, ocular involvement in, 701.
Ascorbic acid, therapeutic, (100), (113), (727), (1078).
and see Vitamin C.
Asmara, trachoma in, (1344).
Association for Research in Ophthalmology, 1941 meeting, 820.
papers read before, **34, 979, 1105, 1115, 1121, 1241, 1255, 1349, 1354.**
Asthenopia, (1218).
Astigmatism, changes in, 1442.
chromatic near test in, (718).
cross-cylinder, evaluation of, 441.
and line-charts, (233), 441.
diagnosis of, by bicylindrical combinations, (717).
torticollis ocularis, atypical, (224).
Astrology and the eye, (846).
Atropine, in glaucoma, (725).
retrobulbar injections of, with diathermy in optic-nerve and retinal disease, (598), (1090).
in retinitis pigmentosa, (1086).

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Australia, Ophthalmological Society of, 1940 transactions, 954.
Automobile drivers, ocular tests for, (846).
Aviators, fitness of, (458).
  naval, physical requirements, 1440.

Bacillus, coli, experimental iridocyclitis from **645.**
  Loeffler, in nonpseudomembranous conjunctival diphtheria, (466).
  Petit, corneal ulcer due to, 446, 447, (966).
Bacterium granulosis, see Trachoma.
Bandage, monocular, (959).
Basedow's disease, see Thyroid disease.
Batavia, carcinoma of meibomian glands in, (1096).
Bee venom in ophthalmology, (1449).
Benzedrine, cycloplegia, see Cycloplegia.
Berg, Frederik, (846).
Berger, rhythm of, (215).
Bergmeister's papilla, (607).
Beta-ray irradiation in ophthalmology, (959).
Biomicroscopy, apparatus, **1433.**
Bitot's spots in Trinidad, **1029.**
Blaskovics's cyclodialysis, (99).
Blennorrhea, adult, see Ophthalmia.
  inclusion, (225), (721), (722), (962).
    corneal involvement in, (1076).
    sulfanilamide treatment of, (964).
Blepharitis, chronic, treatment, (590).
      aniline-dye compound, (107).
      brilliant green, (836).
      eucalyptus, (837).
      naphthalene, (837).
  squamous, castor oil for, (600).
Blepharochalasis, thyroid disease, and harelip, (478).
Blepharoplasty, see Lids.
Blepharoptosis, see Lids.
Blindness, campaign against, in Dutch East Indies, (606).
  causes of, in Hawaii, (1102).
    in Louisiana, (846).
    in Minnesota, (847).
  color, see Color blindness.
  cortical, from cerebral vascular lesion, (1091).
    following pneumothorax insufflation and ventriculography, (833).
  after hematemesis, (1090).
  night, see Nyctalopia.
  ocular developmental anomalies in, (350).
  prevention of, ophthalmology's place in, (116).
    from ophthalmia neonatorum, (607).
    school as educational element in, (1344).
  simulated, (1448).
  statistics relating to, (605).
  sudden, due to acute optic neuritis, (832).
  temporary, from sulfathiazole, (1329).
  of Tobit, (846).
Blind spot, calcium and pilocarpine iontophoresis for enlarged, (1081), (1471).
Blood, -aqueous barrier, (233).
    permeability of, to potassium, sodium, and chloride, (96).
Blood, (*continued*).
  dyscrasias, cerebral symptoms and choked disc in, **1374.**
  glucose in plasma of, (836).
  sulfapyridine distribution in, **60.**
  sulfathiazole concentration in, (957).
  titre in phlyctenular disease, (962).
  vitamin-A content of, in adult gonorrheal conjunctivitis, (91).
  vitamin-C content, in cataractous eyes, (1466).
Blood pressure, see Retinal blood vessels.
Blood transfusion, bilateral spasm of central artery after, (828).
  examination of fundus during, (360).
  in ocular disease, (958).
  for wood-alcohol poisoning, (1470).
Body fluids, see Fluids.
Boeck's sarcoid, of lacrimal gland, (108), **675.**
  of palpebral conjunctiva, (225).
  uveoparotid fever and, (231).
Bolivia, keratoconjunctival lesions in, **900.**
Books—
  American Ophthalmological Society, 1940 transactions, 586.
  Bartley, S. H. Vision—A study of its basis, 1446.
  Bothman, L., and Crowe, S. J., editors. The 1940 year book of the eye, ear, nose and throat, 212.
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  Ophthalmological Society of Australia, 1940 transactions, 954.
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  Snell, Albert C. Medicolegal ophthalmology, 211.

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Books, (*continued*).
Spaeth, Edmund B. The principles and practice of ophthalmologic surgery, 954.
Sverdlick, Jose. Conos y bastoncitos (Cones and rods), 821.
Williams, Jesse Peiring. Personal hygiene applied, 1326.
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Border rays in ophthalmology, (218).
Bourneville's disease, **731.**
Bowmans' membrane, see Cornea.
Brachydactyly with congenital globular lens, **247,** 944.
Brain, hemorrhage, preretinal hemorrhage in, (824).
tumors, metamorphopsia, etc., with, (1091).
ocular symptoms from, **544,** 947, (961).
ten years after removal, 942.
suprasellar, diagnosis of, (105).
visual-field defects with, (834).
Brazil, ophthalmology in, 1066.
Brilliant green, therapy for blepharitis, (836).
Brucellosis, angioscotoma due to, 342.
ocular, 704, (969).
Burns, see Injuries.

Calcium deficiency, lens opacities with, experimental, **611.**
Capsulotomy and iridocapsulotomy, keratome for, **915.**
Carcinoma, of choroid, metastatic from breast, (353), (478), (1335).
from rectum, (840).
in frog, effect of temperature on growth of, (109).
heterotransplantation of, (110).
of iris, metastatic, 81, (355).
of lacrimal sac, (479).
of lids, 74.
of meibomian glands, (1096).
nevo-, of plica semilunaris, (225).
of orbit, metastatic, (353), (1094).
extension from malar region, (355).
of retina, metastatic from breast, **63.**
Cardiovascular system, significance of ophthalmoscopy in diagnosis of, (827).
Carotid artery, internal, aneurysm, (105).
calcified, with optic atrophy, (347).
rupture of, (1330).
Carter, Robert Brudenell, (1343).
Castor oil for squamous blepharitis, (600).
Cataract, allergic eczema and, (973).
biochemistry of, cholesterol and lipid, **191.**
proteolytic activity of, (1227).
capsular, posterior, (975).
congenital, annular, (974).
and oxycephaly, (238).
diabetic, (474).
dinitrophenol, (1227).
dystrophia myotonica, (473).
experimental, in amblystoma-tigrinum, (1085).
dinitrophenol, **680.**
tetanic nutritional, (726).
extraction, (100), (238), (1228), 1443.

Cataract (*continued*).
extraction (*continued*).
aftercare, (1468).
in ambulatory patient? (1227).
bilateral, for senile cataract, 207.
capsulotomy and section, simultaneous, (972).
complicated, prevention, (1085), (1467).
postoperative, (1468).
detachment of choroid, (237), (1228).
hemorrhage, K-avitaminosis in, (1449).
hyphema, **920.**
sympathetic ophthalmia, (232).
death following, (597).
dislocated into anterior chamber, (343).
after glaucoma operation, 1197.
gout, healing in atypical, (100).
intracapsular, (727), (973), (1084).
chi-square test of significance in, (597).
comparison of results from, (237).
forceps for, **689.**
loop for, (597).
simple, 70.
suction, (343), (473), (1468).
tip for Dimitry syringe, (1084).
zonule behavior in, (726).
without iridectomy, (474).
and the occasional operator, (1227).
simple, with keratome incision (Duverger), (726).
sutures to replace fixation forceps, (1466).
vitreous, zonule, and ciliary processes in relation to, (827).
familial hereditary, (1466).
glaucoma with, (1082), (1227).
iritis and, 696.
incipient, dilated iris vessel with, 330.
Kuhnt-Junius choroiditis with, 1314.
stenopeic slit in, (343).
from injury, see Injury.
iris atrophy and high myopia with, 559.
multiple lamellar, (726).
nitrogenous factors anticataractogenic? (473).
rare forms of, (1085).
retinitis pigmentosa and optic atrophy with, 559.
in rickets, adult, 1318.
secondary, due to lens, (1084).
frequency of, (974).
senile, in albino, **798.**
drought as factor in development of, **310.**
extraction in ambulatory patient? (1227).
medication for, thyroxin, (1084).
sex in incidence in, (727).
tetany, (236).
treatment, vitamin C, (100), (727).
vitamin C, (238), (1466).
zonular, sutural and, (1228).
and see Lens.
Central nervous system, diseases of, and eye, (361).

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Chaga's disease, (844).
Chalazion, false, (836), (837).
  nature of, (837).
Chancre of lid, (351).
Chiasm, arachnoiditis, **119,** (476), (832).
  blood-vessel structure of, (475).
  relation to hypophysis, (847).
  syndrome, see Syndrome.
  and see Hypophysis.
Chicago Ophthalmological Society, 325, 588, 694, 943, 1311.
Chickenpox, ocular complications of, (93).
Children, school, examination of eyes of, (606), **779, 1170.**
  rural, ophthalmic conditions among, (1344).
  trachomatous, school for, (361).
Chinese, riboflavin deficiency in, (1100).
Chloroma of orbit, (1096).
Cholesterin, crystals, on the fundus, (101).
  new connective tissue around, (233).
  deposits in the eye, (106), (1460).
Cholesterol content of lens, **191.**
Choline, bodies as pressure-lowering collyria, (219).
  in vascular disease, 1437.
Chorioretinitis, in neuritis papulosa (Fuchs), (976).
  juxtapapillaris (Jensen), with secondary glaucoma, 562, **1429.**
  traumatic, see Injuries.
Choroid, arteriosclerosis of, 325.
  atrophy of, gyrate, **657,** (970).
    with hypogenitalism, (595).
  coloboma of, 697.
    total, (474).
  detachment, after cataract extraction, (237), 330.
  vessels of, pigmented, in Siegrist syndrome, (725).
Choroideremia, (96).
Choroiditis, Kuhnt-Junius, 1314.
  tuberculous, vitreous changes in, (976).
  and see Retinochoroiditis.
Cibazol, see Sulfanilamide.
Cilia, see Eyelashes.
Ciliary-body prolapse with lens dislocation, (1467).
Ciliary margins, vitamin-A ointment for epithelization of, (219).
Cinematography of eye, (715).
Clinics, aniseikonia, 708.
  European, Kuban, perforating ocular injuries in, (358).
Coats's, disease, see Retinitis.
  white rings of cornea, (227).
Cod-liver oil, for desensitizing, (958).
  local application in, keratomalacia, (468).
  restoration of conjunctival sac, (466).
College of Physicians of Philadelphia, Section on Ophthalmology, 75, 335, 561, 946, 1434.
Coloboma, 698.
  of choroid, 698.
    bilateral congenital, 697.
  of iris, (223), (232), 329, 698, (1081).
  of lens, (473).
  of lid, **186,** 203.
Coloboma, (*continued*).
  of optic nerve, in human embryo, **395.**
  of retina and choroid, total, (474).
  of scleral-limbus zone, **615.**
Color curve, see Monkey.
Color blindness, contrast sensitivity in, (1219).
  familial, (717).
  tests of, Ishihara, (1217).
    Rabkin, (960), (1216), (1217).
    Stilling, (1217).
  total, 580.
  types of, (1218).
Color fields, see Visual fields.
Color photography, see Photography.
Color vision, see Vision.
  and see Perception.
Colorado Ophthalmological Society, 74, 329, 701.
Compensation rights for second injuries, (606).
Conjunctiva, (89), (224), (464), (590), (720), (962), (1074), (1222), (1454).
  bulbar, bilharzial granuloma of, (1454).
  chemosis of, from trichinosis, (465).
  cyst of, (1467).
  degeneration of, cholesterin, (1460).
    lipoid, (1460).
  epithelium of, intracellular bodies of, see Inclusion bodies.
  erythema-nodosum involvement of, (465).
  graft, aminoplastin as, (1332).
  leptotrichosis of, (964).
  lesions of, in erythema multiforme bullosum, (224).
  lines on bulbar, (227).
  pemphigus of, see Pemphigus.
  rhinosporidium of, (89).
  as shock tissue in allergy, (1093).
  sterilization of, before surgery, (715).
  tarsal, hyperkeratosis of, (591).
    in early trachoma, (1074).
  tubercle of, benign, (1222).
  tuberculosis of, see Tuberculosis.
  tumors of, see Tumors.
Conjunctival flap in ocular surgery, (219).
Conjunctival sac, restoration of, cod-liver oil in, (466).
Conjunctivitis, allergic, (1074).
  diphtherial, (1074).
    without pseudomembranes, (466), (963).
  in erythema exudativum multiforme, (90), (963).
  Koch-Weeks, (465), (1456).
  monilial, **1307.**
  Parinaud's, (964).
    leptotrich staining in, (458).
    lymphogranuloma venereum with, (721), (835).
    and tuberculosis of conjunctiva, (1455).
  phlyctenular, (226), (962).
  pneumococcus, followed by pneumococcus meningitis, 1434.
  rhinogenous, (466).
  silver colloidal protein for, (1455).
  stenosing, Mooren's ulcer with, (963).
  trachomatous, see Trachoma.

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Conjunctivitis, (*continued*).
treatment of chronic, (590).
vernal, bilateral total degeneration of cornea in, (467).
light etiology for? (1222).
treatment, nicotinic acid, (1223).
Contact glasses, see Spectacle lenses.
Contrast sensitivity, (1217), (1219).
Convergence, (719).
deficiency, **1044.**
measurement of angle of, pocket rule for, (959).
paralysis of, (962).
preservation of, in extraocular-muscle paralysis, **544,** (961).
significance of, in refraction, 560.
and stereopsis in relation to perspective, (222).
in various angles of fixation, (1221).
weakness, (463).
Convocaine, (724).
Corectopia, bilateral, congenital, (471).
Cornea and sclera, (91), (227), (467), (592), (722), (965), (1076), (1224), (1456).
Cornea, allergy of, see Allergy.
in ariboflavinosis, see Keratitis.
astigmatism of, cautery for, (93).
blood staining of, (968), (1077).
Bowman's membrane, (94).
cellular life of, after high temperatures, (1458).
chemistry of, (231).
glucose in, (836).
mucopolysaccharide acid and "spreading factor," (93).
Coats's white rings of, (227).
conical, see Keratoconus.
cyst of, (1460).
in cystine disease, (1456).
degeneration of, (1224).
bilateral, total, in vernal catarrh, (467).
cholesterin, (1460).
familial, (1457).
lipoid, (1460).
Descemet's membrane, (94).
diseases of, pathology, (723).
dystrophy, 450.
calcarious lamellar, (724).
epithelial, (230).
hereditary, (723).
nodular, (227).
ectasia of, (965).
edema of, (1076).
elasticity, anisotropism in, (95).
epithelium, regenerative properties of, (594).
erosions of, recurrent, (1460).
in experimental anemia, (1337).
fistula of, with keratoplasty, 449.
fungus disease of, (1224).
grafts, see transplantation.
hematoma of, (593), (596).
herpes of, see Herpes.
hydrops of, complicating keratoconus, (94).
ichthyosis involving, (95).
inflammation of, ascorbic acid in, (1078).
in chickenpox, (93).
Cornea, (*continued*).
insensitive, Tovell eye shield for, (93).
Krukenberg's spindle, **687.**
leucomata, sensitivity of, (468).
macrocornea, see Macrocornea.
megalocornea, see Megalocornea.
microcornea, see Microcornea.
molding, for contact glasses, (227).
molluscum contagiosum of, (1460).
opacity, in Alaskan Eskimo, (468).
anatomo-pathologic basis of, (1458).
band-shaped, (1461).
sensitivity of, (468).
pannus, Denig's transplant, biomicroscopy of, (229).
placenta for transplant, (1332).
and see Trachoma.
pathological conditions of, (94).
pigs', mechanical properties of, (722).
resection of, 939.
scars, and myopia, (724).
repair of, 1441.
sensitivity of, to Frey's hair, effect of convocaine on, (724).
substantia propria of, (1459).
sulfapyridine distribution in, **60.**
trachomatous, see Trachoma.
transplantation of, (92), (95), (228), (1224), (1457).
cadaver, (968), (1077), (1079).
for central corneal fistula, (1458).
dried and frozen, (1077).
fixation of transplant in vitreous loss, (594).
formolized, (92).
experimental, **1384.**
histologic studies of, (1077).
intracorneal cavity in a, (724).
pathologic anatomy of, (594).
postoperative course in, (230).
restoring vision, (968).
and see Keratoplasty.
tumors of, see Tumors.
ulcers of, (91).
due to Bacillus of Petit, 446, 447, (966).
experimental, effect of typhoid-H antibody concentration on, (92).
and lymphogranuloma venereum, **161,** 335.
Mooren's, (963), (967).
treatment, sulfanilamide, (91), (724).
vitamin, (91).
X-ray, (91).
from yperite, see Injuries.
Corneoconjunctival knife, **322.**
Correspondence, 214, 713, 822.
Cross cylinder, see Refraction.
Crouzon's disease, see Dysostosis.
Cryptophthalmos (ablepharia), (107).
Crystalline lens, (100), (236), (473), (596), (726), (972), (1083), (1227), (1466).
and see Lens.
Csapody tubes, (1459).
Cushing's disease, (844).
Cyanopsia, (1330).
Cyclitis cured after tonsillectomy, (1226).
Cyclodialysis, see Glaucoma.

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Cycloplegia, homatropine, synergistic role of Benzedrine and paredrine in, **282,** 1315.
principle in Datura arborea, (1213).
Cyst, anterior chamber, 329.
of caruncle, (1456).
conjunctival, (1467).
corneoscleral, (1460).
of iris, (1226).
Cysticercus, intraocular, (603), (604).
Cystine disease, ocular findings in, (1456).

Dacryoadenitis, (353), (478).
Dacryocystitis, (1094).
of newborn, (837).
treatment of, chemotherapy, (721).
in newborn, (477).
Dacryocystorhinostomy, burrs for, (478).
Dupuy-Dutemps and Bourget, (352).
suturing sac and nasal mucous membrane in, (107).
Toti's, (351).
Dacryo-sialo-adenopathy atrophicans, see Keratitis sicca.
Daltonism, see Color blindness.
Dark adaptation, see Adaptation.
Datura arborea, mydriatic and cycloplegic principle in, (1213).
Deaths—
Abshire, Joseph T., 1230.
Addleman, Irving Medbury, 729.
Aschenbrenner, Carl, 728.
Ayres, Charles Carlin, 363.
Bailey, Frederick William, 245.
Baird, Robert James, 245.
Ball, Joseph Harris, 1347.
Banner, Allen Carithers, 482.
Barker, Olin G. A., 1104.
Bell, John, 1230.
Bellows, George E., 1230.
Benham, Charles Edwin, 728.
Berrisford, Paul Dee, 977.
Best, Oliver Fletcher, 1473.
Biggs, George Maitland, 1473.
Blackwell, Karl Sigismund, 363.
Bobb, Clarence Curtin, 363.
Boileau, John D., 482.
Breene, Lawrence N., 1230.
Briggs, Francis William, 245.
Brock, George Simeon, 728.
Brown, James Andrew, 245.
Brown, Louis Emmitt, 363.
Bruner, William Thomas, 1104.
Cameron, William Gooderham, 117.
Carleton, Ralph, 117.
Case, William L., 1230.
Caterson, William E., 977.
Chamberlain, Charles T., 608.
Chavasse, Francis Bernard, 1347.
Clark, Ollie P., 728.
Clay, Andrew Jackson, 1104.
Cole, Irving D., 1473.
Connolly, William, 1104.
Conroy, Thomas Francis, 1230.
Copeland, Stanley Earl, 1104.
Coulter, Charles Francis, 608.
Crowder, Thomas W., 848.
Cruickshank, George Robert, 245.
Daly, Joseph Madison, 245.
Deaths, (*continued*).
Davis, Achilles Edward, 608.
Davis, Minot Flagg, 1347.
Dean, Louis Charles, 245.
Dixon, Charles David, 608.
Duncan, John W., 1230.
Dye, John Calvin, 728.
Dyott, Gaven Crane, 1347.
Earel, Fred E., 728.
Ebert, Edwin Charles, 245.
Ellingwood, William A., 1473.
Engel, Harry Perry, 1347.
Ewing, Ulysses B. G., 482.
Farley, Olin Everett, 1473.
Ferguson, Edmund S., 1230.
Ferris, Edgar S., 848.
Franz, Ernest, 1230.
Fridge, John Russell, 728.
Fyke, Will Foster, 363.
Goyer, Thomas Earl, 1230.
Graves, Leonard Knight, 245.
Grimshaw, Robert, 848.
Haggett, Walter Marion, 1347.
Hamaker, William Bernhart, 1230.
Hanger, Franklin McCue, 1104.
Harper, Washington A., 608.
Haseltine, Burton, 1230.
Hawley, Clark Wilder, 245.
Heck, John Joseph, 1104.
Hess, George J. 245.
Hicks, Wesley D., 728.
Hill, Emory, 245.
Hill, J. Frederick, 977.
Hinkle, William Marshall, 245.
Holm, Edgar, 848.
Huizinga, John Gerrit, 363.
Irwin, Frank Newton, 245.
Johnson, John J., 1230.
Jolly, William James, 245.
Joyes, Crittenden, 728.
Kalling, Harold, 977.
Koobs, Herman J. G., 117.
Laws, Joseph H., 848.
Le Moyne, Mederic, 848.
Lewert, Philip John, 1347.
Liebrecht, Emil F. R., 848.
Lockhart, George D., 1104.
Lovell, David Bigelow, 117.
McCarthy, George W., 1104.
MacIsaac, John L., 848.
McPherson, Joseph B., 848.
Mason, Eustace Cosmo, 1230.
Matheny, Ralph C., 977.
May, George William, 245.
Mioton, Sidney Fitzhugh, 728.
Monaghan, Frank Joseph, 1347.
Moody, Thomas C., 848.
Moore, William Alexander, 1104.
Moss, Robert Everett, 1104.
Mulcahy, Thomas A., 848.
Mullen, Thomas R., 1104.
Neff, Charles C., 848.
Niess, Leonard, 608.
O'Brien, Thomas Aloysius, 1104.
O'Brien, William M., 728.
Oliver, Lawrence Henry, 245.
Owen, William Edward, 245.
Page, Rosario J., 482.

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Deaths, (*continued*).
Parr, Lee E., 977.
Pattée, James J., 848.
Peters, Amos Morris, 1230.
Potter, George Benjamin, 608.
Quinby, William O'Gorman, 848.
Reckard, Hiram Leslie, 1230.
Renninger, Abner R., 1230.
Rhoads, Harmon Talley, 728.
Ritter, Howard Murray, 1230.
Rogers, Robert Merriam, 977.
Rosenthal, Joseph Morris, 1473.
Roth, William Fritchey, 977.
Russell, Hiram B., 728.
Sackrider, George P., 848.
Sackrider, John Raymond, 1473.
Sanderson, Herman Harvey, 1230.
Sawyer, Walter Wesley, 245.
Scales, Herbert Livingston, 977.
Scott, Merle E., 1104.
Seright, Thomas C., 1104.
Shine, Francis Wayles, 1473.
Sickel, Edward Wilson, 1104.
Simmons, Ralph Hayward, 848.
Smith, Edgar Francis, 977.
Smith, Eugene, 1104.
Sorell, Frank William, 728.
Stephenson, Eugene T., 977.
Strickler, David A., 848.
Swan, Tyrus Eugene, 1230.
Talmage, John Burr, 482.
Taylor, Lillian Ethel, 117.
Teehan, George Edward, 608.
Thomson, William Johnstone, 245.
Tiffany, DeForrest Elmer, 977.
Tomkins, George Johnson, 977.
Touchstone, Alexander Green, 1347.
Tracy, John M., 1230.
Trimble, Charles Elmore, 363.
Van Wagner, Lee Cone, 1230.
Wadley, Samuel Lynn, 363.
Walker, Claude William, 1347.
Waltz, Manford Repp, 977.
Warren, Edmund L., 728.
Warwick, Harold L., 977.
Watts, Felton D., 1230.
Westover, Robert Leland, 1347.
White, Joseph Augustus, 608.
White, Peter Cope, 1104.
Wills, Leon Clifford, 363.
Wolfe, Claude Thomas, 848.
Woodruff, George F., 728.
Woodruff, Thomas A., 848.
Wootton, Herbert Wright, 728.
Yazujian, Dikran M., 977.
Zimmerman, George Abram, 1473.
Denig's plastic operation, see Trachoma.
Depth perception, see Perception.
Dermoids, corneoconjunctival, 698.
Descemet's membrane, see Cornea.
Desensitization, with adrenalin, (219).
to tuberculin, see Tuberculin.
Deseptyl (p-aminobenzolsulphamid), in inclusion-body affections of genital origin, (226).
myopia from ingestion of, (1218).
Diabetes, ocular complications of, (1341).
cataract, (474).
Diabetes, (*continued*).
fundus, (825), (1100), (1470).
retinal veins, (244), 570.
retrobulbar neuritis, (348).
with retinitis proliferans, 1311.
Diagnosis, general methods of, (215), (458), (714), (957), (1211), (1447).
Diathermy, effect of, on anterior segment, (459).
on eye, (217).
and iontophoresis for glaucoma, (1083).
in ophthalmology, (217), (220).
in optic-nerve and retinal disease, (598).
for retinal, hemorrhage, (244).
tumor, (1094).
Weve electrode, modification of, **322.**
and see Retinal detachment.
Dictyoma, (1336).
early, (1095).
Dimitry suction syringe, tip for, (1084).
Dinitrophenol cataract, see Cataract.
Diphtheria of the eye, (1074).
Diplopia, charting of, 569.
monocular, **503.**
nasopharyngeal tumors and, (833).
Doryl, effect of, pressure-lowering, (219).
Drusen of optic nerve, see Optic nerve.
Dutch East Indies, campaign against blindness in, (606).
Dysostosis, craniofacialis (Crouzon), hereditary, **1014.**
multiplex (Hurler), and eye symptoms, (107).
and Morquio's disease, (1101).

Editorials—
A.M.A. meeting, Section on Ophthalmology, 1941, 820.
American Academy of Ophthalmology and Otolarngology, 1941 meeting, 1445.
American Ophthalmological Society, 1941 meeting, 819.
Association for Research in Ophthalmology, 1941 meeting, 820.
Bilateral cataract operation, 207.
Change of editorship, The, 79.
Changes in astigmatism, 1442.
Dinner for Dr. Post, The, 84.
Doryl in glaucoma, 1444.
Exophthalmic ophthalmoplegia, 455.
Expansion of aniseikonic clinics, 708.
Familial macular dystrophies, 82.
Fundus reflexes, 705.
Gentle art of reading a medical journal, The, 1069.
Glaucoma survey, 1206.
Gonioscopy in glaucoma, 1323.
Heredity in refraction, 816.
Industrial ophthalmology, 584.
Iseikonic enthusiasm, 1321.
Learning ophthalmology, 1320.
Metastatic carcinoma of the iris—diagnosis, 81.
Ocular rheumatism, 706.
Office practice, 456.
Optics in the Middle Ages, 337.
Ophthalmology in Brazil, 1066.

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Editorials, (*continued*).
Pacific Coast Oto-Ophthalmological Society, 1941 meeting, 818.
Pan-American board of ophthalmology, A, 80.
Personal health, 815.
Prognosis in uveal sarcoma, 1064.
Realistic orthoptic reports, 582.
Selective draft and medical preparedness, The, 210.
Spring ophthalmological meetings, The, 819.
So-called compensated glaucoma, 83.
Sulfanilamide compounds in ophthalmology, 339.
Sulfanilamide locally for gonorrheal ophthalmia, 453.
Total color-blindness, 580.
Two problems, 951.
Tuberculin reaction in ophthalmology, 1067.
Uveal disease, 1063.
Visual zone, The, 579.
Vitamin therapy in ophthalmology, 1325.
War and ophthalmic literature, 711.
What type of cataract extraction? 1443.
William Mackenzie, 1204.
Editorship, change in, 79.
Einthoven, phenomenon of, and length of living eye, (106).
Elastometer, ballistic, (236).
Electrocardiographic alterations in essential hypertension, (102).
Electroencephalography, eye movements in, (463).
in ocular affections, (458).
Elliot's operation, see Glaucoma.
Embolism of central retinal artery, see Retinal blood vessels.
Emphysema, orbito-palpebral, traumatic, (599).
Encephalopathy, lead, choked discs in, **794.**
Enophthalmos, and orbital tumor, (1094).
traumatic, see Injuries.
Entropion, see Lids.
Entoptic phenomena, (960).
in polarized light, (460).
Epinephrine, role of, in formation of intraocular fluid, **1105.**
Epiphora, canaliculorhinostomy for, (1332).
hypophyseal adenoma and, (1332).
Jameson operation for, 944.
muco-conjunctival canal for, (838).
Episcleritis, see Scleritis.
Epithelioma, of ciliary retina, (840).
of conjunctiva, (1337).
palpebral, (1334).
of lid, irradiation treatment of, (354).
of limbus, (1334).
meibomian, (351).
Erythema, bullosum (Stevens-Johnson disease) involving eyes, (224).
multiforme exudativa, ocular involvement, 568.
with conjunctivitis and stomatitis, (90), (963).
nodosum, involving conjunctiva, (465).
Eserine and pilocarpine, mechanism of, (1463).
Eskimo, Alaskan, corneal opacities in, (468).
Eucalyptus for blepharitis, (837).
Evipal-sodium, see Anesthesia.
Evisceration, (349).
combination curet and suction spoon for, (1449).
with scleral implant and preservation of cornea, 71.
Exenteration, stump for prothesis, (1331).
Exophthalmos, (1092).
experimental, (476), (834).
in hyperthyroidism, **527.**
first symptom of, **1423.**
malignant, after thyroidectomy, (106).
pulsating, (1330).
treatment, (1092).
surgical, (1330).
from tumor, (353), (355).
unilateral, (477), 695, (835).
pathogenesis and treatment of, (1092).
from tumor, (353).
Eye, allergy of, **759.**
anatomy of, 587.
astrology and the, (846).
cast-making of living, (1459).
diseases of, blood-sedimentation rate in, (1212).
gonococcal, (604).
lysozyme in, (460).
malarial, intravenous salt solution to demonstrate, (1212).
mast cells in, (835).
in Moscow, (1343).
occupational, see Injuries.
oxygen therapy of, (1450).
scrofulous, carbohydrate metabolism in, (1100).
sulfonamides in, see Sulfonamides.
in Ydmurt, (1345).
and ear inorientation and balance, (116).
effect of, diathermy on, (217).
prolonged administration of thyroid on, (1329).
exchange of, between Amblystoma punctatum and Triturus viridescens, (362).
function and structure of, **277,** (599).
hospitals, see Eye hospitals.
infections of, see Infections.
inflammation of, see Inflammations.
influence of, on human psyche and physical make-up, (116).
injuries of, see Injuries.
innervation of, sympathetic and parasympathetic, (1345).
length of living, Einthoven phenomenon and, (106).
mercurochrome as antiseptic for, (714).
microtomy of, (217).
optimum working conditions for, (114).
parasites in, see Parasites.
pathology, classifications of, (606).
pregnancy and, see Pregnancy.
relation of central-nervous-system disease and, (361).
size of blind spot after water-drinking test, (1466).
and sleep, (1091).
Eyeball and orbit, (106), (349), (476), (599), (834), (1092), (1330), (1472).

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Eyeball, curvature of, (1459).
displacement of, operation, (223).
elasticity, measurement of, (236).
transplantation of, (1330).
wax modeling of, (361).
Eye clinics, see Clinics.
Eye, ear, nose and throat specialists, 822.
Eye grounds, see Fundus.
Eye hospitals, Kalinin, corneal transplantation in, (592).
Madras, trachoma data from, (467).
Talsk, isothermic box in, (459).
Eyelashes, trichiasis, Van Millingen's knife for, (352).
Eyelids and lacrimal apparatus, (107), (351), (477), (600), (836), (1093), (1332).
Eyelids, see Lids.
Eye shields, Tovell, (93).

Fibrohemangioma of orbit, transcranial extirpation of, 75, (355).
Fibroma of orbit, (110).
Fibrosis of lacrimal caruncle, (226).
Fields, see Visual fields.
Filaria, see Parasites.
Flicker, phrynosoma response to, (1103).
visual, critical intensities for, (1450).
Fluids, ocular, (96).
distribution of sulfanilamide in, (959).
role of epinephrine in formation of, **1105.**
Focal infection, teeth, retinitis and superior-rectus paresis in, (598).
and see Nasal accessory sinuses.
Forceps, epilation, (836).
for intracapsular-cataract extraction, **689.**
Foreign bodies, see Injuries.
Fovea, effect of dark objects in visual field on differentiation sensitivity of, (1215).
of vertebrates, comparative histophysiology of, (362).
Fuchs, gyrate atrophy of choroid and retina, **657,** (976).
neuritis papulosa, (976).
Fundus, autofundoscopy, **1403.**
changes in, in diabetes, (825), (1100), (1470).
in experimental anemia, (1337).
inflammatory, differential diagnosis, (1468).
in old injury, by sodium light, (1098).
in optic-nerve atrophy seen by sodium light, (1090).
in pituitary disease seen by sodium light, (1090).
following splanchnectomy in malignant hypertension, (829).
cholesterin crystals on, (101).
in Cushing's disease, (844).
cysticercus in, (603), (604).
hemorrhages, after sulfanilamide, (714).
in hypertension, clinical, (102).
essential, (102).
malignant, (829).
in functional psychosis after insulin-shock therapy, **26.**
reflexes, 705.
photography of, see Photography.
vascular affections of, 560.
Fungus, aspergilli, etc., in corneal disease, (1224).
rhinosporidium, (89), (720).
Fusion, (720).

Gases, see Injuries.
Gaucher's disease, biopsy of typical pingueculae, (464).
Giza Memorial Ophthalmic Laboratory, 13th annual report, 341.
Glass blowers, xerosis of cornea in, (593).
Glasses, see Spectacle lenses.
Glaucoma and ocular tension, (99), (234), (472), (596), (725), (970), (1081), (1226), (1462).
Glaucoma, 1194.
acute, cataract with, (1227).
effect of intravenous sodium salicylate in, (235).
infiltration of stellate ganglion in, (234).
mechanical factors in etiology of, **851.**
pathogenesis of, 1201.
treatment of, 1198.
biomicroscopy of anterior segment in, and choice of surgery, (1464).
capsular, central lens capsule in, (970).
exfoliation of anterior lens capsule in, (1081), (1467).
cataract with, (1082).
dislocated, (343).
and iritis, 696.
chronic, simple, 1313.
"compensated," 83.
definition of, 1195.
with drusen of optic disc, **1138.**
effect of, darkness on blind spot and angioscotoma in, (234).
pilocarpine on light sensitivity of, (1464).
elasticity of eyeball in, (236).
elastotonometric curve in, (1463).
electroencephalography in, (458).
gonioscopy and, (1083), 1323.
in prognosis and treatment, **1396.**
hemorrhagic, (345).
hereditary tendency to, (472).
iridocyclitis and hypopyon with, (1462).
juvenile, 695.
hereditary, (235).
liver-function test in, (1082).
malignant, 1195.
metabolic studies of, (235).
following mydriasis, **668,** (970).
and nevus flammeus, (1463).
pathogenesis, of acute, 1201.
of simple, 1202.
primary, hereditary, (472).
prodromal, adaptation test in early, (1462).
retinitis pigmentosa with, (1470).
secondary, histopathology of, 564.
Jensen's chorioretinitis and, 561.
size of blind spot in, after water-drinking test, (1466).
spherophakia with, **247,** 944.
sugar-tolerance curves in, (1465).
survey of, 1206.
tension, changes in, in experimental anemia, **(1337).**

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Glaucoma, (*continued*).
tension, (*continued*).
effect of, choline bodies as collyria on, (219).
eserine and pilocarpine on, (1463).
furfuryl trimethyl ammonium iodide, (235).
iontophoresis on, (236).
Meckel's ganglion on, (971).
muscular exercises on, (1081).
normal intraocular pressure in therapy, (472).
pituitary extracts on, (972).
peripheral innervation of the iris on, (596).
hypertension, pupillomotor drugs in, (234).
hypotension, papilledema in, (105).
during insulin coma, **1174.**
mechanism of, in mammalia, (1463).
nerve pathways for regulation of, (1465).
tolerance, (971).
value of graphs of, 1200.
and see Abstracts, section eight.
traumatic, see Injuries.
treatment, of acute, 1198.
high-frequency currents, (472).
medical, atropine, (725).
doryl, 1444.
splenic extract, (725).
surgical, (1082).
aftercare, (1468).
choice of, (234), **768,** (970), (971), 1199, (1464).
dark-adaptation after, (971).
cyclodialysis, anterior combined, (1227).
gonioscopic observations after, **1026.**
inverted (Blaskovics's), (99), (472).
dialysis, conjunctival, for recurrent, **788.**
diathermy and calcium ionization of the neck, (1083).
effect of, on eye, 1203.
iridencleisis, corneal hematoma after, (596).
iridocorneosclerectomy, curved knife for, **804.**
iridotorsion, (234).
optic-atrophy, progressive after, 1196.
scleral incision for approach to shallow anterior chamber, (235).
sclerocorneal trephining (Elliot's), (472).
drill trephine, (596).
late results, (596).
trephine sharpening, (235).
value of gonioscopy in, **1392.**
uveitis with, 702.
and see Abstracts, section eight.
Glioma; **1354.**
of retina, bilateral, (838).
heredity in, (478).
prognosis, (355).
treatment, diathermy coagulation, (839).
radium, (838).
Glucose in avascular ocular tissues, (836).
Gonioscopy, binocular, (1211).
Gonioscopy, glaucoma and, (1083), 1323, **1392.**
Gonorrheal ophthalmia, see Ophthalmia.
Graves's disease, see Thyroid disease.
Grenfell, Sir Wilfred Thomason, **86.**
Grenz rays, see Border rays.
Groenouw's corneal dystrophy, see Keratitis.
Grosseteste, Robert, on optics, (114).
Gumma, carcinoma of iris simulating, (355).

Haidinger's polarization brushes and ocular defects, (460).
Hamblin-Dallós contact lens, see Spectacle lenses.
Harada's disease, (98), (1462).
Hawaii, causes of blindness in (1102).
Head injuries, see Injuries.
Heart size, radiological, in essential hypertension, (102).
Heerfordt's uveoparotitis, see Syndrome.
Heine's cyclodialysis, see Glaucoma.
Helmholtz, see Ophthalmoscope.
Hematemesis, blindness following, (1090).
Hematology, see Blood.
Hematoma, corneal, (593), (596).
of orbit, from hemophilia, (599).
Hemeralopia, see Nyctalopia.
Hemianopsia, bitemporal, traumatic, (348).
cortical, (1091).
after head injuries, (1472).
homonymous, (348), 1313.
from retinal arteritis, (349).
Wollaston and, **1053.**
Hemophilia, hematoma of orbit in, (599).
Hemorrhage, orbital, spontaneous, (1330).
postoperative, K-avitaminosis in? (1449).
preretinal, (824), 1311.
retinal, in internal leishmaniosis, (1228).
in newborn, (598), (1469).
in cases of subarachnoid hemorrhage, (1469).
subconjunctival, blood staining of cornea from, (968).
Heparin therapy for retinal-vein thrombosis, (598).
Hepatic disease, glaucoma and, (1082).
Kayser-Fleischer ring with, 559.
Heredity in ophthalmology, (1344).
Herpes zoster ophthalmicus, ocular changes in, (226).
experimental, (1459).
sector corneal lesions in, (965).
treatment, convalescent blood, (459).
ultraviolet, (722).
Heterochromia of iris, 200.
Heterophoria, asthenopia and, (1218).
measurement of, (1220).
tests of, (961).
High-frequency currents in ophthalmology, (958).
Hill, Emory, **212.**
Hippel's diease, see Angiomatosis retinae.
Hirschprung's disease, see Megacolon.
Hissar, nightblindness in, (240).
Histamine sensitivity, (459).
History, see Ophthalmology.
and see Astracts, section eighteen.
Holth's iridencleisis, see Glaucoma.

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Homatropine, cycloplegia, see Cycloplegia.
solubility of, (958).
Hordeola, see Lids.
Hormones, testicular, in treatment of myopia, (88).
Hospitals, see Eye hospitals.
Hotz operation for entropion, see Lids.
Hyaloid artery, development of, (607).
persistent, (607), (1081).
Hyaloid membrane, persistent, (1087).
Hydrocephalus, with megacolon and optic atrophy, (348).
Hydrophthalmos, 696.
supposed relation with macrocornea, (1225).
Hygiene, personal, 1326.
Hygiene, sociology, education, and history, (114), (361), (605), (846), (1101), (1343).
Hypertelorism, (349), 567, **928.**
with cleft palate and giant-cell tumor, (350).
Hypertension, aneurysmal dilations of retinal arteries in, (239).
arterial, 325, (604).
obliteration of central retinal artery in (824).
clinical, the fundus in, (102), (103).
essential, fundus changes in, (102), (829).
and peripheral optic pathways, (105).
ocular, see Glaucoma and ocular tension.
Hyperthyroidism, see Thyroid disease.
Hypertropia, occlusion, (1074).
Hyphema after cataract extraction, **920.**
Hypogenitalism, gyrate atrophy of choroid and retina in, (595).
Hypophysis, ocular symptoms from, in Basedow's disease, (349).
tumors of, 949.
adenomata, 947, (1332).
ocular symptoms from, syndrome of cavernous sinus, (476).
Hypopyon, with iridocyclitis and glaucoma, (1462).
keratitis, (229), (843).
Hypotony, ocular, see Glaucoma and ocular tension.

Ichthyosis, ocular, (95).
Illumination, functional, in college, (1343).
rational, (847).
standardization of Snellen chart, **287.**
trans-, (216).
and see Light.
Illuminator, focal, **439.**
Implantation, plastic material for, **550.**
polyviol, (1448), (1449).
vitallium, (1092).
scleral, 71.
Inclusion blennorrhea, see Blennorrhea.
Inclusion body in trachoma, see Trachoma.
Indelible-pencil injury, see Injuries.
Industry, concept of visual performance in, (1102).
injuries in, see Injuries.
Infections, fungus, see Fungus.
ocular, gonococcic, see Ophthalmia.
pneumococcic, (721).
sulfonamides in cases of, (1448).
Infections, (*continued*).
and see Focal infections.
and see Nasal accessory sinuses.
Inflammations, in inherited syphilis, (605).
of posterior pole, (1468).
rheumatic, of the eye, (1342).
Infrared rays, injury of cornea and lens from, experimental, (111).
Injuries, (111), (355), (479), (601), (841), (1097), (1337).
alabaster, cement, and sand, experimental, (1340).
anterior chamber, foreign body in, blackberry thorn, (1097).
glass membrane in, (843).
xeroform ointment in, (843).
anterior segment, penetrating, (358).
asphyxia, traumatic, with optic atrophy, 808.
bee sting, hypopyon keratitis from, (843).
bullet wounds of occipital region, (359).
burns, chemical, (480), (842).
lye, experimental, (1098).
hot sand, (357).
treatment, (601).
cod-liver oil, (1099).
surgical, (356).
cataract, electric, (1338).
from foregin body, (112).
traumatic, (479), 566.
operative technique for, **803.**
chemical, (359).
chorioretinitis, central, traumatic, 559.
compensation for, see Compensation rights.
conjunctival, sulfathiazole reactions, (112).
contusions, fundus lesion from, (1338).
hole in macula from, (355).
transitory pupillary change, (1337).
of cornea, bee sting, (843).
birth, (843).
burns, (601).
irradiation, (111).
dystrophy, from yperite, (841), (843).
edema, traumatic, (842).
perforating, (843).
ulcer, from gold salts, (841).
from yperite, (843).
xerosis of, in glass blowers, (593).
enophthalmus, traumatic, (1098), (1341).
of eye, (1345).
cephalic tetanus following, **1281.**
juvenile, results, (1340).
nonindustrial, (1339).
penetrating, 1438.
perforating, (358), (843), (1097), (1098).
rupture of posterior ciliary arteries, (358).
war, (480).
of eyelids, see lids.
foreign body, in anterior chamber, (356).
in cornea, (842).
neuroretinitis after removal, (1339).
experimental, glass, (842).
intraocular, (602), (842), 1441.
cataract formation from, (112).
late extraction of, (1339).
localization of, (480), (1097).
diascleral illumination, (1339).

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Injuries, (*continued*).
foreign body, (*continued*).
intraocular, (*continued*).
localization, (*continued*).
scleral projection, (1339).
Trendelenburg's stereoroentgenography, (359).
Vogt's, (355).
X-ray, (1340).
magnetic, (602).
electro-, (1097).
magnet, (602).
arcing eliminated in footswitch of, (479).
of iron-nickel-aluminum alloy, (1098).
removal, 330.
anterior route, late results, (359).
diascleral, (603), (1340).
prophylaxis against retinal detachment, (1098).
posterior route, **553.**
steel, retained and dilated pupils, 330.
nonmagnetic, copper, spontaneous expulsion, (358).
glass, (843), (1338).
removal, (357), (480).
from ciliary body, (1099).
gases, mustard, (357).
experimental, ascorbic-acid effect on, (113).
glaucoma, chronic, due to shock, (970).
head, bullet wounds, (359).
cerebral metamorphopsia after, (1472).
hemianopsia from, (348).
syndrome of third visual neuron, (1471).
indelible pencil, (602).
industrial, 584, (593), (841), (1343).
legal medicine and, (606).
prophylaxis from pitch injury, (601).
from intolerance of gold salts, (841).
iridocyclitis, alopecia from, (601).
iridodialysis, traumatic, (602).
surgical repair of, 943.
keratoconus and, (228).
lacrimal-gland luxation, (1098).
of lacrimal sac, traumatic rupture, (113).
of lens, 70.
irradiation, (111).
from trauma, 566.
and see cataract.
of lids, treatment, (1099), 1440.
macular, hole, (355), 588, 697.
from metrazol treatment, (112).
myopia, from radium irradiation, (1339).
from sulfanilamide, see Sulfanilamide.
from nupercaine, (113).
ophthalmia nodosa, hairy moss, (357).
of orbit, (1099).
aniline pencil, (602).
firearm, X-ray diagnosis of, (1338).
fractures, (601).
pitch, (601).
of retina, angiopathy, traumatic, (357), (358), **537.**
artery, with cerebral fat embolism, (356).
degeneration of, experimental pigmentary, (602).
Injuries, (*continued*).
of retina, (*continued*).
detachment, (240), (241).
from indirect trauma, (344).
saponin, (111).
of sclera, rupture, (1097), (1340).
sella turcica, fracture of, with disturbance of hypophyseal function, (358).
sphenoidal-fissure syndrome after, (356).
sulfathiazole, (112).
vitreous detachment, (1097).
war, (480).
gases, (357).
penetrating, 1438.
yperite, (841), (843).
Insects, color vision in, (847).
Instruments, sharp, sterilization of, 206, **624,** (959).
Insulin-shock treatment, ocular changes in **26, 1174.**
Interstitial keratitis, see Keratitis.
Intraocular fluids, see Fluids.
Intraocular pressure, see Glaucoma.
Iontophoresis, calcium, for enlarged blind spot, (1081), (1471).
for glaucoma, (1083).
effect of, on ocular tension, (236).
quinine bisulphate, for recurrent trachoma after sulfanilamide treatment, (225).
Irgamid, see Sulfanilamide.
Iridencleisis, see Glaucoma.
Iridocapsulotomy, scissors for, **915.**
Iridocyclitis, with Boeck's sarcoid of lacrimal gland, **675.**
experimental, from bacteria in upper respiratory tract of man, **645.** (969).
with hypopyon and glaucoma, (1462).
tuberculous, X-ray treatment of, (970).
Iris, aniridia, congenital, (471).
atrophy of, high myopia and cataract with, 559.
changes in syphilis, (1462).
coloboma, (223), (232), 329, 698, (1081).
cyst of, spontaneous, (1226).
dilator, cholinergic behavior of, **34.**
heterochromia of, 200.
peripheral innervation of, and intraocular pressure, (596).
prolapse, trichloracetic acid for, (1214).
pseudomelanomas of, **133.**
structure of, elastic, (1080).
tumors of, see Tumors.
vessel, dilated, with incipient cataract, 330.
Iritis, acute, from sulfamide and vaccine treatment of blennorrhea, (99).
cataract and glaucoma with, 696.
nodular, (98).
recurrent, (99).
rheumatic, 706, (1342).
tuberculous, see Tuberculosis.
Iseikonic enthusiasm, 1321.
Ishihara color plates, see Color plates.

Jameson operation for epiphora, 944.
Jaw-winking, see Syndrome.
Jensen's retinochoroiditis juxtapapillaris, 561, **1429.**

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Kalinin Eye Hospital, corneal transplantation in, (592).
Kayser-Fleischer ring, hepato-lenticular degeneration, 559.
Keloid of cornea, (601).
Keratitis, allergic, experimental, effect of thyroidectomy and thyroxin on, (592).
in ariboflavinosis, (481), (1079).
band-shaped, 447.
and uveitis in polyarthritis, 701.
bullous, experimental, (1457).
deep pustuliform, (722).
disciform, 443.
vaccinal, (94).
epidemic, (230).
and see Keratoconjunctivitis.
herpetic, see Herpes.
hypopyon, (229), (843).
in inclusion conjunctivitis of newborn, (1076).
interstitial, (966).
sector, in herpes zoster ophthalmicus, (965).
in syphilis, and allergy, (966).
congenital, (837).
tuberculous, Rubrophen treatment, (1226).
from mumps, **655.**
neuroparalytic, (1079).
after coagulation of gasserian ganglion, (1459).
nodular (Groenouw's), (93), (227).
punctate, superficial, in Malaya, (1226).
in Tasmania, (592).
rheumatic, 706, (1342).
role of trigeminus in, (1459).
subepithelial leprous, (92).
treatment, surgical, transplantation of corneal tissue, (1225).
ulcerative, (1224).
Keratoconjunctivitis, in Bolivia, **900.**
epidemic, (228), (229), (469), (1224), (1225).
punctate superficial, (92).
scrofulous, seasonal effect on, (592).
sicca, (838), (968).
in vitamin-E-free dieted rats, (1100).
Keratoconus, (88).
hydrops of cornea complicating, (94).
and injury, (228).
relation of avitaminosis A and D to, 214.
treatment, cautery, (93).
surgical, (468).
Keratoderma, (95).
Keratoglobus, (88), 451.
Keratoleptynsis, dystrophic marginal, with cavity, (468).
Keratomalacia, cod-liver oil as local application in, (468).
local action of oils containing vitamin A, (94).
from vitamin-A deficiency, (966), (1225).
xerophthalmia with, and bile deficiency, (1225).
Keratome, **915.**
Keratoplasty, **1,** (92), **139.**
Filatov's, modified, (228).
fistula of cornea with, 449, (1457).
Keratoplasty, (*continued*).
with formalinized transplants, (92).
at Kalinin Eye Hospital, (592).
late results, (593).
and see Cornea.
Knife, corneoconjunctival, **322.**
ophthalmic, **1309.**
sclerectomy, curved, **804.**
for Van Millingen grafting, (352).
Koch-Weeks bacillus, see Bacillus.
Krukenberg spindle, **687.**
Kuhnt-Junius retinopathy, 1314.

Lacquer in ophthalmology, (224).
Lacrima, absence of, (965).
antibacterial action of, lysozyme, (1094).
on staphylococci, **635.**
Lacrimal apparatus, (1093).
function of, capillary test of, (600).
pathology of, treatment, (1093).
surgery, (352), (353).
Lacrimal canaliculi, anomaly of, (108).
repair of, 941.
stenosis of, (353).
streptothrix infection of, **682.**
Lacrimal caruncle, fibrosis of, (226).
retention cyst of, (1456).
supernumerary, (1075).
Lacrimal glands, dacryoadenitis, (352).
diseases involving, (838).
hypofunction of, **20,** 698.
sarcoid of, see Tumors.
Lacrimal sac, dacryocystitis, see Dacryocystitis.
extirpation of, (1332).
granuloma of, (108).
oncocyte hyperplasia of, (1093).
phlegmon, X rav for, (1333).
tumors of, see Tumors.
Lagrange operation, see Glaucoma.
Lamina cribrosa, blood supply of, (1103).
Laurence-Moon-Biedl syndrome, see Syndrome.
Leber's disease, see Optic-nerve atrophy.
Leishmaniasis, in eyelid, (1093).
retinal hemorrhage in, (1228).
Lens, abnormal, congenital, (1467).
secondary glaucoma due to, (1084).
absorption of, effect of diathermy on, (973).
anomalies at posterior pole, (1097).
biochemistry of, 70, **979,** (1083).
carbohydrate, role of calcium in metabolism of, (974).
cholesterol, **191.**
glucose, (836).
lipid, **191.**
seasonal variations in, (973).
melanoidin formation, (237).
phosphates, acid-soluble, (596).
oxidation of fatty acids of surviving, (972).
proteolytic activity of, (1227).
vitamins, 69.
C, in cataractous and normal eyes, (1466).
capsule, coronal striations in, (1085).
exfoliation of, (1081), (1467).
coloboma of, (473).

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Lens, (*continued*).
diffusion of dyes in, (343).
dislocation of, and accommodation, **40.**
adherent to optic disc, (1468).
anterior, 206.
in arachnodactyly, see Marfan's syndrome.
as isolated factor in, (474).
congenital, bilateral, 701.
with prolapse of ciliary body, (1467).
separation of zonular lamella from, 694.
and see Injuries.
ectopia, see dislocation.
effect of, calcium deficiency on, (236).
hydrogenation of naphthalin on, (237).
extraction, in high myopia, **519,** (972).
without iridectomy, (474).
globular, anterior, developed at puberty, (974).
congenital, with brachydactyly, **247.**
injuries, see Injuries.
lesions in eye contusions and perforations, see Injuries.
opacities of, adrenal induced, (239), (974).
and experimental calcium deficiency, **611.**
experimental, (1083).
origin of, by induction in salamander, (238).
persistent vascular tunic of, (975).
pigmentation of, posterior, **687.**
reaction of, to infrared rays, (111).
and vitamins, 69.
zonular layer, anatomy of, (1103).
and see Cataract; and see Abstracts, section nine.
Lenses, see Spectacle lenses.
Leprosy, ocular involvement of, (92), (1454).
Leptotrich staining, (458).
Leslie Dana award to Dr. Arnold Knapp, 849.
Leucoma, amelioration of, (595).
experimental, (1080).
Leukemia, ocular changes in, (1341).
Lids, anomaly of, 698.
border disease, ultraviolet irradiation for, (722).
in Chagas's disease, (844).
chancre of, (351).
coloboma of, **186,** 203.
ectropion, cicatricial, effect of testicular extract on, (1333).
surgical repair of, 944.
from meibomian epithelioma, (351).
operation for, (108).
emphysema of, following fracture of frontal sinus, (351)).
entropion, spastic, 204, (352).
treatment, surgical, (1093).
trachomatous, treatment, **298,** (837), (1332).
insertion of levator palpebrae muscle, (600), **749.**
leishmaniasis, (1093).
Marcus Gunn syndrome, see Syndrome.
plastic surgery of, (1333).
placenta for, (1332).
restoration, 204
of upper, **46.**
Lids, (*continued*).
ptosis, operations for, (600).
Eversbusch, (600).
relation of orbicularis and levator, (837).
as shock tissues in allergy, (1093).
sporotrichosis of, (1332).
style, false, (836).
synkinesis of, (837).
syphilitic involvement of, (351), (837).
tarsus, see Tarsus.
tumors of, see Tumors.
upper, unilateral spasmodic retraction of, (600).
Light, polarized, (460).
entoptic phenomena in, (460).
ocular phenomena produced by, (1216).
sense, effect of vitamins on, (1219).
sensitivity, effect of pilocarpine on, in glaucoma, (1464).
sodium, macular lesions seen by (216), (827), (1090), (1098).
ophthalmoscopy by, (957).
in pituitary disease, (1090).
Limbus, leproma of, (1454).
tumors of, see Tumors.
Liver disturbances, see Hepatic disease.
Lizard (phrynosoma), flicker response in, (1103).
Localization, visual, neural mechanisms of, **1255.**
Los Angeles Society for Ophthalmology and Otolaryngology, 1440.
Louisiana, blindness in, (846).
Lucite, diagnostic contact lens of, (215).
for implantation, **550.**
Lupus erythematosus, ocular changes in, (230).
Lymphadenosis, ocular tumor with, (600).
Lymphoblastoma of orbit, (1334).
Lymphocytoma, ocular, (354).
Lymphogranuloma venereum, ocular, (721), (835).
corneal ulcer with, **161,** 335.
Lymphogranulomatosis, ocular complications of, (838).
Lymphoma of conjunctiva, (109).
Lysozyme, (1094).
in ocular disease, (460), **635.**

Mackenzie, William, 1204.
Macrocornea and hydrophthalmus, (1225).
Macula lutea, changes of, unilateral, 1312.
degeneration of, disciform, 697, (828).
juvenile, 702.
Kuhnt-Junius, 559.
dystrophy, familial, 82, (103).
hole in, central, inflammatory, 1311.
traumatic, see Injuries.
initial lesions of, seen with sodium light. (216), (827), (1090), (1098).
lesions of, 1315.
of monkey, demonstration of an absorption color curve in, (116).
Maddox-rod test, **156.**
Magnet, see Injuries.
Maklakov tonometer, (1226).
Malaria, ocular symptoms in, (603).

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Malaya, epidemic superficial punctate keratitis in, (1226).
Malingering, (1345).
Marcus Gunn syndrome, see Syndrome.
Marfan's syndrome, see Syndrome.
Mariotte, blind spot of, (1211).
Marx-Schmidt drinking test, see Glaucoma.
Mast cells in ocular pathology, (835).
Mecholyl, effect of, on glaucoma, (219).
Meckel's ganglion, relation of, to accommodation and intraocular pressure, (971).
Medical journal, art of reading, 1069.
Megalocornea, Krukenberg's spindle with, **687.**
  and sporadic megalocornea globosa, (1225).
Megocolon, optic atrophy with, (348).
Melanocarcinoma of choroid, (1096).
Melanoma of ciliary body, benign, (109).
Melanosarcoma, of choroid, (346), 807, (1334).
  of eyeball, (838).
  of orbit, (109).
  of retina, diathermy treatment of, (1094).
  of uvea, (111), (840).
Melanosis, of conjunctiva, malignant change in, 808.
  of eye, (838).
    congenital, (98).
      histology, (354).
Meningitis, pneumococcus, following pneumococcic conjunctivitis, 1434.
Menstruation, scotoma associated with, **507,** (833).
Mercurochrome as antiseptic in eye, (714).
Mercury, oxycyanide, for trachomatous pannus, (966).
  yellow oxide of, bactericidal action of, (1214).
Metals, magnetic properties of various, (602).
Metamorphopsia, due to brain tumor, (1091).
  after head injury, (1472).
Microcephaly, congenital retinal folds and, (825).
Microcornea, (223).
Microphthalmos, unilateral, with tumorlike neuroglial proliferation, 1436.
Microscopy, fluorescence, of ocular tissues, (1447).
Microtomy of eye, (217).
Migraine, ophthalmic, (89), 812, (844).
  ophthalmoplegic, with iris and aqueous involvement, (1220).
Mikulicz's disease, (838).
Minnesota Academy of Ophthalmology and Otolaryngology, Section on Ophthalmology, 71, 703.
Minnesota, causes of blindness in, (847).
Miosis, see Pupils.
Molluscum contagiosum of cornea, (1460).
Mongols, luetic ocular diseases among, (360).
Monkey, absorption color curve in macula of, (116).
Morquio's disease, ocular findings in, (1101).
Moscow, ocular diseases in, (1343).
Mucopolysaccharide acid of cornea, relation to "spreading factor," (93).
Mucous-membrane transplantation, (91).
  and see Trachoma.
Mumps, keratitis due to, **655.**
  optic neuritis after, (830).
Muscles, balance, at reading distance, (1453).
    routine test of, (464), 703.
    screen test of, **156.**
  disturbances of, in neuro-ophthalmologic conditions, 813.
  effect of alcohol on, (599).
  extraocular, 712.
    disturbance of motility in hyperthyroidism, (1452).
    in myasthenia gravis and hyperthyroidism, 75.
    neuromuscular transmission in, (1345).
  injuries of, see Injuries.
  insertions of superior oblique and retinal tears, (826).
  intrinsic, analogy of, with heart muscle, (471).
    pharmacologic behavior of, **34,** (222).
  oculomotor-nerve paralysis, in nasopharyngeal tumors, (833).
  operations on, for absence of inferior rectus, (223), (224).
    advancement, (224).
    tendon transplantation for paralysis of external rectus, (462).
    transposition of, in monkeys, **1115.**
  paralysis, of abducens, see Abducens.
    of abduction, with retraction of eyeball, (1220).
    diplopia charting in, 569.
    divergence, **556.**
    of external muscles, preservation of convergence with, **544.**
      rectus, following dental infection, (598).
        following spinal anesthesia, (462), (719).
        tendon transplantation for, (462).
    surgical treatment of, (1073).
Mydriatics, glaucoma following the use of, **668.**
  platyphyllin, (715), (716).
  principle in Datura arborea, (1213).
Myasthenia gravis, external ophthalmoplegia in, 75.
Myoma of ciliary body, (110).
Myopia, (718).
  avitaminosis and, **408.**
  corneal scars and, (724).
  development and prevention in U. S. Naval Academy, (1102).
  high, iris atrophy and cataracts with, 559.
    lens extraction in, **519,** (972).
    osmotherapy, (1217).
  Lindner theory of, criticized, (223).
  origin of, (1451).
  progressive, keratoconus and keratoglobus, (88).
  temporary, from sulfanilamide, (217), **323,** (461), 567, (715), **895, 935,** (1218), (1219).
  treatment by testicular hormones, (88).

Naphthalene for blepharitis, (837).

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Nasal accessory sinuses, ocular affections from, (350), (599).
fracture of frontal, lid emphysema after, (351).
optic-tract lesions, (1228).
retinal detachment, (1228).
pathology of, effect of operative windows on, 703.
tumors of, see Tumors.
and see Focal infections.
Nasopharynx, malignancies of, ocular symptoms from, (833).
Nematode worm in anterior chamber, (113).
Nerves, ciliary, in the human, (1346).
pathways, principal, 1207.
third, regeneration of, and ocular motility, (1461).
sixth, see Abducens.
Neuralgias, facial, tuberculin therapy, 86.
Neurinoma of orbit, (1337).
Neurofibroma of oculomotor nerve, (1334).
Neurofibromatosis (Recklinghausen's), involving ocular structures, with choroidal neoplasm, (1337).
of lids, (479).
with orbital neurinoma, (1337).
retinal, (478).
surgical treatment of, 1437.
and tumors of optic nerve, **1144.**
Neuromyelitis optica, 200, (347).
Neuromyoarterial glomus tumor of eyelid, (839).
Neuro-ophthalmological subjects, 200, 813, 823.
Nevus flammeus and glaucoma, (1463).
New England Ophthalmological Society, 72, 200, 331, 452, 704, 949.
New York Eye and Ear Infirmary, Ophthalmological Department, 446.
New York Society for Clinical Ophthalmology, 67, 69, 203, 441, 566, 811, 1316, 1438.
News Items, 117, 245, 363, 482, 608, 728, 848, 977, 1104, 1230, 1347, 1473.
Night blindness, see Nyctalopia.
Nyctalopia, determination of, (242), (1328).
experimental, color field in, (1451).
functional, etiology and treatment, (1468).
in Hissar, (240).
Nystagmus, miner's, (719).
dynamics of, (1453).
in Netherlands coal fields, (962).

Obituaries—
Grenfell, Sir Wilfred Thomason, 86.
Hill, Emory, 212.
White, Joseph A., 1208.
Wolfe, Claude Thomas, 955.
Woodruff, Thomas Adams, 1209.
Wootton, Herbert Wright, 1072.
Occlusion by laquered lens, (224).
Ocular infections, see Infections.
Ocular movements, (89), (223), (462), **485, 619,** (719), **741,** (961), (1073), (1219), (1451).
optic-nerve traction during, (1087).
and see Muscles.
Oculogyric crises, **1000.**
Odessa Medical Institute, electromagnetic operations in, (1097).
Office practice, (115), 456.
Ointments, container for ophthalmic, **933.**
ophthalmic, bactericidal properties of, (1214).
Onchocercosis, see Parasites.
Operations, ocular, (218), (605).
conjunctival flap at limbus in, (219).
delirium after, (459).
and see Surgery; and see Abstracts, section two; and see also under individual subjects, such as Cataract, Glaucoma, Retina, or the personal name associated with the particular operation.
Ophthalmia, gonorrheal, (604).
bilateral, **690.**
relation of adult, to vitamin-A content of blood, (91).
treatment, **879.**
sulfamides, (99), (721), (1076), (1448), (1449), (1454), (1455).
local application, **314,** 453.
neonatorum, prevention of blindness from, (607).
prophylaxis, (1222).
ampoules for silver-nitrate solution, (227).
therapy, **879.**
sulfanilamide, (464).
local application, (466).
nodosa, see Injuries.
sulfonamide treatment of, (1456).
sympathetic, see Sympathetic ophthalmia.
Ophthalmodynamometry of retinal circulation, (1089).
Ophthalmoeikonometer measurements, repeatability of, (461).
Ophthalmological Societies—
American Academy of Ophthalmology and Otolaryngology, 1941 meeting, 1445.
American Medical Association, Section on Ophthalmology, 1941 meeting, 820.
American Ophthalmological Society, 1941 meeting, 819.
Chicago, 325, 558, 694, 943, 1311.
Colorado, 74, 329, 701.
Los Angeles, 1440.
Minnesota, 71, 703.
Netherlands, 99 meetings, (114).
New England, 72, 200, 331, 452, 704, 949.
New York Eye and Ear Infirmary, Ophthalmological Department, 446.
New York Society for Clinical Ophthalmology, 67, 69, 203, 441, 566, 811, 1194, 1316, 1438.
Ophthalmological Society of Australia, 1940 meeting, 954.
Philadelphia, 75, 335, 561, 946, 1434.
Pacific Coast Oto-Ophthalmological Society, 1940 meeting, 457; 1941 meeting, 818.
Research, 820; and see Association for Research on Ophthalmology.
Royal, 570, 806, 1318.
Saint Louis, 206, 443, 939, 1437.
Ophthalmologists and occultists, (846).
and personal health, 815.
thoughts on office practice, (115), 456.

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Ophthalmology, bee venom in, (1449).
in Brazil, 1066.
centers in America, (605).
heredity in, (1344).
history of, fifty years in the U.S. (1101).
industrial, 584.
lacquer in, (224).
learning, 1320.
leprosy in, see Leprosy.
literature, war and, 711.
in medicine, (1344).
medico-legal, 211.
moulage in, (361).
preventive, (116).
sulfanilamide compounds in, see Sulfonamides and Sulfanilamide.
textbooks of, 585, 1071.
tuberculin reaction in, 1067.
value of yellow light in, (216).
vitamin therapy in, see Vitamins.
in wartime, (115), (1102).
and see Abstracts, section eighteen.
Ophthalmoplegia, exophthalmic, 455, (961).
external, complete, preservation of elevation of eye to corneal stimulus in, (463).
in myasthenia gravis, 75.
in nasopharyngeal tumor, (833).
Ophthalmoscope, binocular, (1212).
Helmholtz, ninety years of, (1343).
Ophthalmoscopy, auto-, **1403.**
diagnosis of cardiovascular disease and, (827).
narrow beam of light for, (1448).
simple method of, (458).
yellow light, (216), (957), (1090), (1098).
Optic agnosias, (220).
Optic chiasm, see Chiasm.
Optic nerve and toxic amblyopias, (104), (346), (475), (598), (830), (976), (1090), (1329), (1470).
Optic-nerve atrophy, calcified carotid artery, cupping, and low tension with, (347).
in exophthalmic goiter, (1091).
eye ground in sodium light in, (1090).
after hematemesis, (1090).
Leber's, (344).
cured by neurosurgery, (347).
marble bones and, **874.**
megacolon and hydrocephalus with, (348).
in pernicious anemia, (1090).
progressive, after glaucoma operation, 1196.
retinitis pigmentosa and, 559.
syphilitic, treatment, **119,** (1471).
retrobulbar atropine sulfate, (1090).
tryparsamide, (348).
traumatic asphyxia with, 808.
Optic nerve, aplasia of, 561.
blood-vessel structure of, (475).
coloboma of, in human embryo, **395.**
degeneration of, from prolonged administration of thyroid, (1329).
disc, Bergmeister's, variations in development and regression of, (607).
choked, from brain tumor, 942.
in cerebral pseudotumor, (360).
Optic nerve, *(continued)*.
disc, *(continued)*.
choked, *(continued)*.
cerebral symptoms and, in blood dyscrasias, **1374.**
diagnosis of, angioscotometry in, (216).
in lead encephalopathy, **794.**
drusen formation in, (1090).
and choroidal melanosarcoma, (346).
glaucoma associated with, **1138.**
related to tuberous sclerosis, (104).
edema of, in ocular hypotony, (105).
disease, and dark adaptation, (1471).
retrobulbar injections of atropine and strychnine with diathermy for, (598).
histologic structure of, (1103).
intracranial, modes of compression of, (347).
traction on, effect of retina during ocular rotation, (1087).
tumors of, see Tumors.
and see Brain tumors.
X-ray examination of, (104).
Optic neuritis, acute, sudden transitory blindness from, (832).
after mumps, (830).
papulosa (Fuchs), (976).
retrobulbar, (104).
choroidal perithelioma simulating, (354).
early sign of pernicious anemia, (347).
sodium-nitrite therapy for, (346).
from tissue anoxia, (831).
toxic, dental infection, (598).
and see Neuroretinitis.
sinus involvement, (1228).
Optic tracts, blood-vessel structure of, (475).
lesions of, electric examination of occipital region in, (215).
peripheral, general hypertension and, (195).
Optico-ciliary neurectomy, (1331).
Optics, Robert Grosseteste writer on, (114), 337.
in the Middle Ages, 337.
Orbit, actinomycosis of, (350).
anatomy of, 587.
aneurysm of, cirsoid, (477).
cellulitis, Ludwig's angina with, after molar extraction, (1472).
cyst of, dermoid, (477).
emphysema, (599).
hematoma of, from hemophilia, (599).
hemorrhage, (1330).
hyperostosis of, **1005.**
implants, see Implantation.
injuries of, see Injuries.
phlegmon of, (1092).
prothesis for, (1331).
pseudotumors of, (477), (599).
bilateral, inflammatory, (1472).
tumors of, see Tumors.
and see Abstracts, section thirteen.
Orbital apex syndrome, see Syndrome.
Orthoptic exercise, (223), **1022,** (1451), (1453).
coöperative ophthalmic laboratory for, **1057.**

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Orthoptic exercise, (*continued*).
home training in, **1299.**
at Milwaukee Children's Hospital, **1019.**
practical, 588.
realistic reports of, 582.
results of, (962).
and surgical correction of strabismus, **1418.**
and see Strabismus.
Osmotherapy for high myopia, (1217).
Osteitis deformans, angioid streaks with, (1469).
Osteomalacia, cataract in, 1318.
Oxycephaly, ocular involvement in, (238).
and see Dysostosis.
Oxygen therapy of eye disease, (1450).

Pacific Coast Oto-Ophthalmological Society, 1940 transactions, 457, 1941 meeting, 818.
P-aminobenzolsulfamid, see Deseptyl.
Pan-American board of ophthalmology, 80.
Pan-American Congress of Ophthalmology, 1940 meeting, papers presented before, **900, 920, 1005, 1144, 1185, 1281, 1392, 1396.**
Pannus, see Cornea; and see Trachoma.
Panophthalmitis, following operation for retinal detachment, (826).
tuberculous, (95), (1331).
Papaverine as a collyrium, (219).
Papilla, see Optic nerve.
Papilledema, see Optic nerve.
Papilloma of conjunctiva, (108).
Pappataci fever, ocular complications of, (361).
Parasites, Bilharzia, (1454).
Filaria, bancrofti, (113).
loa, (361).
onchocercosis, (481), **1185.**
Schizotrypanum cruzi, (844).
and see Abstracts, section seventeen.
Paratrachoma, (722).
Paredrine, solubility of, (958).
and see Cycloplegia.
Parinaud's conjunctivitis, see Conjunctivitis.
Parotitis, epidemic, ocular involvement, (1099).
Pemphigus, ocular, 450, (963), (1222).
Pentothal sodium anesthesia, (1215).
Perception, color, anisochromatopsia, congenital, (88).
and see Abstracts, section three.
depth, meaning and measurement of, 811.
modified Perlia test, for middle distance, (221).
in relation to perspective, (222).
Perithelioma of choroid, (354).
Perlia test, see Perception.
Petit, diplobacillus of, see Bacillus.
Phakoma, retinal, (478), (1335).
Philadelphia College of Physicians, see College of Physicians of Philadelphia, Section on Ophthalmology.
Phlyctenulosis, blood titre in, (962).
carbohydrate metabolism in, (1075).
Phorias, see Strabismus.
Phorometer and accommodation rule combined, (1219).
Photography, of fundus, (216).
color, (216).
Phototherapy, see Ultraviolet.
Physiologic optics, refraction, and color vision, (88), (220), (460), (716), (959), (1215), (1450).
Physiologic optics and the patient, 442.
Pilocarpine, effect of, on intraocular pressure, (1463).
on light sensitivity in glaucoma, (1464).
Pituitary, disease, see Hypophysis.
extracts, effect of, on tension and pupil, (972).
Plasmocide, effect of prolonged therapy on eye, (1213).
ocular disease from, therapy of, (1330).
Plastic surgery, see Surgery.
and see under the various tissues and adnexa.
Platyphyllin, mydriatic properties of, (715), (716).
Pneumothorax insufflation, followed by blindness, (833).
Post, Lawrence T., dinner for, 84.
Pregnancy, ocular involvement in, (1341).
Prisms of thermoplastic material, (221).
Proctocain anesthesia, see Anesthesia.
Proptosis, see Exophthalmos.
Prostate, carcinoma of, metastatic to eye, (353).
Prothesis, individually molded, (106).
orbital, (1331).
for plastic operations (porcelain), (716).
stump for, and optico-ciliary neurectomy, (1331).
Prowazek-Halberstaedter bodies, see Inclusion bodies.
Psammoma of orbit, **664,** (1095).
Pseudopemphigus, (1222).
Pseudotumor, cerebro-pituitary origin of, (360).
of orbit, (477), (599), (1472).
pseudoglioma of retina, **164.**
Pseudoxanthoma elasticum and angioid streaks of the retina, see Retina.
Pterygium, (91), **790.**
histopathology of, (1222).
operation for, (91), (465), (591), **790,** (1455).
mucous-membrane grafts, (91).
Ptosis, see Lids.
Pupil, Argyll Robertson, (969), (1461).
corectopia, bilateral, congenital, (471).
dilation of, centers of, (1345).
from retained steel, 331.
effect of pituitary extracts on, (972).
miosis, congenital, (99).
pupillometer, see Pupillometer.
reaction of, efferent pathway of, (724).
size of, in experimental anemia, (1337).
tonic, (969).
site of disturbance in, (595).
in syphilis, (1462).
Pupillary membrane, persistent, with other malformations, (232), (471).
Pupillometer, entoptic, **1431.**
Purkinje figure of Walker, **1403.**
Purtscher's traumatic retinal angiopathy, **537.**

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Quinine amblyopia, (346).
in children, (346).

Rabkin's polychromatic plates, see Color blindness.
Radiography, see X rays.
Radium treatment, for glioma retinae, (838).
for palpebral epithelioma, (354).
transitory myopia from, (1339).
Rayleigh's interval, effect of adrenalin on, (222).
Rebates, 951.
Recklinghausen's disease, see Neurofibromatosis.
Refraction, **640,** (960).
abduction and adduction ratio and, 1438.
accommodation and convergence in, significance of, 560.
astigmatism, bicylindrical-combination test of, (717).
crossed-cylinder and line-chart tests of, **(223),** 441.
contact-glass, 443, (717).
cycloplegia in, see Cycloplegia.
cross-cylinder, (222), 441, (461).
rotary, **438.**
simultaneous comparison and, 822.
errors of, in twins, (1219).
heredity in, 816.
muscle-balance test in, (464), 703.
retinoscopy, (1218).
sensitometric, (1217).
significance of Sturm's interval in, **413.**
skiascopy, 579, 713.
and see Abstracts, section three.
Respirators, optical appliance for use with, (960).
Reticuloma of conjunctiva, (1336).
Retina and vitreous, (101), (239), (344), (474), (597), (824), (975), (1085), (1228), (1328), (1468).
Retina, absence of, and of choroid, (474).
angioid streaks of, (1087), (1229), (1328).
with osteitis deformans, (1469).
with pseudoxanthoma elasticum, (345).
angiomata of, (240).
atrophy of, gyrate, **657,** (976).
in hypogenitalism, (595).
changes in, angiospastic, 1312.
diabetes, see Diabetes.
seen with sodium light, (216), (828), (1090), (1098).
circulation in, ophthalmodynamometry in study of, (1089).
degeneration of, experimental, (598).
hereditary, familial, (240).
Leber's (344).
from prolonged administration of thyroid, (1329).
diseases of, (830).
edema of, (1470).
and change in electric retinal sensitivity, (1089).
effect of optic-nerve traction in ocular rotation on, (1087).
external limiting membrane, relation of, to Verhoeff's membrane, (1103).
Retina, (*continued*).
folds of, congenital, and microcephaly, (825).
hemorrhage in, diathermy for, (244).
in internal leishmaniasis, (1228).
in newborn, (598), (1469).
preretinal, (824), 1311.
in subarachnoid hemorrhage, (1469).
lesions of, from indirect trauma, see Injuries.
macula, see Macula lutea.
neuroepithelium of, structure and histochemistry of, (1345).
periphlebitis, tuberculous etiology of, (830).
photography of, see Photography.
pigment, migration of, (1219).
pigmentation of, grouped, 560.
rents in, see Retinal detachment.
rods and cones, 821.
striae, after detachment, (826).
structure of, (605), (1345).
technique of surgery, (829).
tonometry of, see Retinal blood vessels.
vascular connective-tissue layer between pigment epithelium of, and lamina vitrea, (1345).
visual purple of, cataphoretic mobility of, (1215).
molecule, weight of chromophore carrier in, (221).
vitamins and, (243).
Retinal blood vessels, anatomy of finer, (1087).
arteries, arteriosclerosis of, 325.
blood pressure in, instability of, from carotid sinus, (244).
central, aneurysmal dilatations of, in hypertension, (239).
embolism of, (830).
acetylcholine in, (975).
obliteration of, in arterial hypertension, (824).
obstruction of, 943.
tension of, in cranioencephalic traumatism, (597).
occlusion of, (345).
spasm of, (241), (345), (828).
superior temporal, embolism of, **936.**
autoretinovasoentoscopy, **1403.**
caliber of, effect of anoxia and high concentration of oxygen on, (102).
changes in essential hypertension, (102), (103).
crossing of, (975).
disease of, X-ray therapy of, (241).
veins, central, pressure in, (1088).
thrombosis, (103).
heparin in, (598).
tubercle obstructing, (345).
changes in, in diabetes with arterial hypertension, (244).
lesions of, cleavage of retina and, (244).
pulsation of, (1087).
thrombosis, (830).
treatment, (1088).
X-ray, (241).

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Retinal detachment, (101), (243), (830).
and accident, (240), (475).
acetylcholine production in, (1086).
adhesion between retina and vitreous in, (824).
in aphakia, (237).
bilateral, in progressive papillo-retinal edema, (825).
electric sensitivity in, (240).
electroencephalography in, (458).
equatorial scleral staphyloma and, cured, **403.**
etiology and pathology of, 74.
holes (rents, tears), closure of posterior, (975).
insertions of superior oblique muscles and, (826).
localization, (1085), (1099).
seen with sodium light, (828).
idiopathic and traumatic, 702.
prophylactic, in threatened separation, (1468).
retinopathia centralis angiospastica and, (1469).
from sarcoma of choroid, (841).
serosa allergica and, (1469).
and sphenoidal sinusitis, (1228).
staphyloma with, excision, **403.**
traumatic, (240), (241), (475), 702.
treatment, operations, diathermy, (101), (1089).
high-frequency currents, (958).
results after, panophthalmitis, (826).
remote, (1229).
at Washington University School of Medicine, 445.
vitreous, zonule, and ciliary processes in relation to, (827).
Retinal periphlebitis, tuberculous, (345).
Retinitis, diabetic, (348), (825).
clinico-pathologic study of, 570.
exudative, Coats's, 562.
senile macular, (102).
hypertensive, (103).
seen with sodium light, (827).
macular, juvenile exudative, 695.
metastatic, (826).
pigmentosa, (830), (1086).
glaucoma with, (1470).
hereditary, (344).
with optic atrophy and cataracts, 599.
pathogenesis of, (830).
peripheral vascular picture in, (474).
treatment of, retrobulbar atropine sulfate, (1086).
proliferans, 1438.
with diabetes mellitus, 1311.
spasm in, (345).
tuberculous, (1342).
and see Neuroretinitis.
Retinoblastoma, see Glioma.
Retinochoroiditis, Jensen's, see Chorioretinitis.
Retinography, see Photography.
Retinoscopy, see Refraction.
Rheumatism, ocular, 706.
and see Arthritis; and see Inflammations.
Rhinosporidium, see Fungus.
Riboflavin, see Vitamin B_2.
Rickets, adult, see Osteomalacia.
Rickettsia in conjunctival epithelium of pigs, (962).
in trachoma, see Trachoma.
Rodent ulcer, see Cornea.
Rods and cones, see Retina.
Roentgen ray, see X ray.
Royal Society of Medicine, Section of Ophthalmology, 570, 806, 1318.
Rubrophen dye for tuberculosis of eye, (220), (1214), (1226).
Rumania, contribution of, to trachoma problem, (1103).

Saint Louis Ophthalmic Society, 206, 443, 939, 1437.
San Francisco orthoptic laboratory, **1057.**
Sante Fé, trachoma campaign in northern, (1102).
São Paulo, trachoma in S. José dos Campos, (1343).
Saponin, injury to the eye, (111).
Sarcoid, Boeck's, see Boeck's sarcoid.
Sarcoma, of choroid, (840).
diagnosed after operation for detachment, (841).
near eye, causing exophthalmos, (355).
sympathetic ophthalmia from nonperforating, (1096).
of uveal tract, (1334).
prognosis, (355), 1064, (1096).
Schlemm's canal, visible, **615.**
Schools, ocular policy for, **779.**
for trachomatous children, (361).
Sclera, abscess, (1460).
blue, (1460).
deficiency of, bilateral, (592).
episclera, lipoid droplets in, (968).
staphyloma, equatorial, and retinal detachment, excision, **403.**
and see Abstracts, section six.
Sclerectomy, see Glaucoma.
Sclerencleisis, see Glaucoma.
Scleritis, anoxia in, (965).
episcleritis, role of anoxia in, (965).
rheumatic, (1342).
Scleroderma and ocular involvement, (106).
Scleroperikeratitis, progressive, pathogenesis, (968).
Sclerosis, multiple, 202.
tuberous, fundus changes in, (1335).
relation of drusen of optic nerve to, (104).
retinal, **731.**
Scotoma, action of calcium and magnesium ions on edemagenic, (1471).
annular, **196.**
associated with menstruation, **507,** (833).
central, (217).
scintillating, prognosis, (359).
Scotometer, (714), (1211).
Screen test, **156,** 812.
Selective draft and medical preparedness, 210.
Sensitivity, light and color, see Perception.
and immunity in experimental ocular tuberculosis, (845).

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Short-wave therapy, effect of, on normal and diseased eyes, (1212).
Shwartzman toxins, ocular reaction to (1341).
Silver, colloidal protein therapy of pneumococcic infections, (1455).
nitrate, for trachomatous pannus, (1458).
prophylaxis, (227), (1222).
salts, effect of, on eye, (1213).
Sinuses, see Nasal accessory sinuses.
Skiascopy, see Refraction.
Sleep, the eye and, (1091).
Slitlamp, epilation forceps for use with, (836).
portable, **1190.**
Snellen chart, see Visual acuity.
Sodium iodate, toxic for retina, (602).
Sodium light, see Light.
Sodium nitrite for retrobulbar neuritis, (346).
Spatula, single and double-ended, **66.**
Spectacle lenses, antiglare, for night driving, (223).
bifocal, (223).
colored, modification of vision by, (716).
contact, (717), 949.
aid in fitting, **439.**
corneal molding for, (227).
diagnostic, of lucite, (215).
gonioscope, (1211).
Hamblin-Dallós, (716).
in refraction, 443.
for straightening eyes, **933.**
lacquered, for obscuring vision, (224).
prescription for, (1217).
prisms of thermoplastic material, (221).
transposition of, **1308.**
for use with respirators, (960).
Spectrum, blue-green region of, (222).
Spherophakia with glaucoma, **247,** 944.
Spielmeyer-Vogt's disease, see Amaurotic idiocy.
Splenic extract for glaucoma, (725).
Sporotrichosis, palpebral, (1332).
Spring catarrh, see Conjunctivitis.
Staphylococcus, antibacterial action of tears on, **635.**
aureus, and panophthalmitis, (826).
Stereopsis, binocular, see depth under Perception.
Stereoscopic pictures, Russian, (1102).
Stevens-Johnson disease, see Erythema multiforme.
Stilling color plates, see Color blindness.
Stomatitis in erythema multiforme, (90), 568.
Strabismus, (720).
angle of, determination of, (462).
convergent, (463), 694.
accommodative, (89).
mirrorlike in twins, (462).
etiopathogenesis of, (961), (1220).
functional, (1453).
treatment, (464), (1221), (1452), (1453).
critique of, (462).
nonoperative, **1022,** (1221).
and operative, **1418.**
orthoptics, (89), (223), **1299,** (1451).
in concomitant strabismus, (962).
Strabismus, (*continued*).
treatment, (*continued*).
nonoperative, (*continued*).
orthoptics, (*continued*).
at Milwaukee Children's Hospital, **1019.**
spectacles of complementary colors, (1220).
operative, (1219), (1221), (1222), **1418.**
prism spectacles after, (464).
for vertical, (462), (1452).
vertical, (462).
and see Muscles.
Streptothrix infection of canaliculus, **682.**
Stye, see Lids.
Sulfanilamide, appearance of, in aqueous, (1449).
compounds in ophthalmology, **292,** 339, **428,** (1214).
and see Sulfonamides.
for corneal leasions, (91).
distribution of, in aqueous, (232).
in ocular fluids and tissues, (959).
effect of, on Koch-Weeks bacillus, (1455).
fundus hemorrhages from, (714).
for inclusion conjunctivitis, (964).
myopia from, acute, transitory, (217), **323,** (461), 567, (715), **895, 935,** (1218), (1219).
for ophthalmia, gonorrheal, (1076), (1454), (1455).
local use in, **314,** 453, (466).
neonatorum, **314.**
sympathetic, **49.**
for trachoma, (90), **174,** (720), (964), **1035,** (1075).
recurrence after, (225).
for vaccinia of the eyes, (108).
Sulfapyridine, distribution of, between blood, aqueous, and cornea, **60.**
for ulcus serpens corneae, (724).
Sulfathiazole, permeation of, in eye, (957).
temporary blindness from, (1329).
Sulfonamide therapy, 339, (1214), (1449), (1456).
cibazol, (1214).
irgamid, (1214).
p-amino benzol-sulfamide (deseptyl), myopia from, (1218), (1219).
septoplyx, (1454).
streptocide, (1448).
tioseptal, (1448).
ultraseptyl, (1454).
Surgery, ocular, 701, 954.
adhesive tape for use after, (959).
celluloid shield for postoperative wounds, (959).
plastic, 205.
embryonal tissue for, (466).
placenta for, (1332).
prothesis for, (716).
problems, (218).
retinal, (829).
sterilization of conjunctiva before, (715).
technique and instruments in, (100).
in World War I, 1439.
and see Operations.
Suśrutan's principles of ocular therapy, (1101).

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Sutures, black and white silk, **1192.**
Symblepharon, surgery of, (465).
Sympathetic ophthalmia, (97), (231).
  after cataract extraction, (232).
  etiology, (1080).
  from nonperforating sarcoma, (1096).
  pathogenesis of, (969).
  therapy for, (1080).
    atophanyl and sodium salicylate, (96).
    sulfanilamide, **49.**
  and see Abstracts, section seven.
Syndrome, Adair-Dighton's, see Van der Hoeve's.
  Adie's, (97), (98), 202, (233), (1461).
  anterior choroid artery, (844).
  baso-frontal, 1312.
  cavernous sinus, (476).
  chiasmatic, (832).
    prechiasmal, (476).
  Gunn's, 806, 808, (1093).
  Hand-Schüller-Christian, 946.
  Harada's, (725), (1462).
  Heerfordt's, 806.
  Laurence-Moon-Biedl, 588.
  Marfan's, (238), (474), (972), (1467).
  nasal ganglion, (226).
  orbital apex and sphenoid-fissure, (350), (356).
  Siegrist, (725).
  Van der Hoeve's, **57,** 700.
Syphilis, hereditary, inflammations of the eye in, (605).
  interstitial keratitic, (837), (966).
  iris changes in, (1462).
  ocular, 67, 72.
    among the Mongols, (360).
  of optic nerve, **119,** (348), (1090), (1471).
  and tonic pupils, (1462).
  treatment, 68.
    tryparsamide, (348).
      with vitamin-B therapy, (219).
Systemic disease and parasites, (113), (359), (481), (603), (844), (1099), (1341).

Tachycardia, paroxysmal, ocular compression in, (1342).
Tape, adhesive, (959).
Targesin in ophthalmology, (1455).
Tarsus, aplasia of, (351).
  turning of, for trachomatous entropion, (837).
Tasmania, superficial punctate keratitis in, (592).
Tay-Sachs disease, see Amaurotic idiocy.
Tears, see Lacrima.
Teeth, see Focal infection.
Teletraumatism, retinal, **537.**
Tension, ocular, see Glaucoma.
Terrien's disease, see Keratoleptynsis.
Testicular extract, effect on cicatricial ectropion, (1333).
Tetanus, cephalic, after injury to eyeball, **1281.**
Therapeutics and operations, (217), (459), (714), (957), (1212), (1448).
Therapy, Suśrutan's principles of, (1101).
  ultrashort wave, (217), (1212).
  ultraviolet, (722).
Thrombosis of retinal vein, (1088).
  development of clinical picture in, (103).
  heparin for, (598).
  X-ray therapy for, (241).
Thymol, action of, and cod-liver oil in desensitizing, (958).
Thyroid disease, blepharochalasis and, (478).
  exophthalmic, **527,** (1330), (1331).
    first symptom of, **1423.**
    malignant, after thyroidectomy, (106).
    optic atrophy in, (1091).
    X-ray treatment of, (349).
  extraocular muscles in, 75, (1452).
Thyroidectomy, effect of, on allergic experimental keratitis, (592).
Thyroid extract, effect of, on dark adaptation, (976).
    prolonged administration on eye, (1329).
Tioseptal, see Sulfonamides.
Tobacco amblyopia, see Amblyopia.
Tobit, blindness of, (846).
Tonometer, Maklakov's (1226).
Tonoscopy, see Retinal blood vessels.
Torticollis ocularis, atypical, (224).
Toti's operation, see Dacryocystorhinostomy.
Trachoma, in Asmara, (1344).
  campaign against, role of visiting nurse in, (607).
    in Rumania, (1102).
    in northern Santa Fé, (1102).
  classification of, (1345).
  cornea, pannus, histology of follicular, (968).
    medication, mercury oxycyanide, (966).
      silver nitrate, (1458).
    surgery, Denig's, (229), (720).
      embryonal tissue for, (466).
      slitlamp picture of reaction to, (464).
  corpuscles of, see rickettsia.
  diagnosis, (1224).
    from cytology of tarsal conjunctiva, (1074).
    doubtful, (1223).
  distribution of, in the United States, (115).
  entropion due to, see Lids.
  etiology of, 1326, (1455).
    Prowazek bodies, (90), (225), 1326.
  experimental data from Madras, (467).
  history of, 1326.
  incidence of, in San José dos Campos, (1343).
  inclusion-body conjunctivitis with, (721).
  involving caruncle, (226).
  pathogenesis of, (90).
  pathology of, (467).
  rickettsia in, (964).
  schools for trachomatous children, (361).
  serologic reactions in, (1455).
  treatment of, antimony, (590), (591).
    chemotherapy, 85, (963).
      deseptyl, (226).
      silver colloidal protein, (1455).
      sulfanilamide, (90), **174,** (720), (964), **1035,** (1075), (1223), (1456).
        recurrence after, (225).
    for military candidates, (590).

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Trachoma, (*continued*).
treatment of, (*continued*).
suction, (1076).
surgical, tarsectomy, (1223).
transplantation of conserved sclera, (591).
and tuberculin allergy, (1454).
virus, Weil-Felix reaction in, (721).
Transfusion, see Blood transfusion.
Transillumination, see Illumination.
Transilluminator, (216).
Transplantation, see Cornea; and see Keratoplasty.
Trephine, drill, (596).
sharpening, (235).
Trephining, see Glaucoma.
Trial case, see Spectacle lenses.
Trichiasis, operation for, placenta for, (1332).
Van Millingen's, (352).
Trichinosis involving conjunctiva, (465).
Trichloracetic acid for iris prolapse, (1214).
Trimethoxy-dioxy-oxotritane, see Rubrophen.
Trinidad, Bitot's spots in, **1029.**
Triturum viridescens, exchange of eyes with Amblystoma punctatum, (362).
Trochlear palsy, surgical treatment of, 565.
Trombeteira (Datura arborea, D. fastulosa L.), mydriatic and cycloplegic principle in, (1213).
Trypanosomiasis, (844).
Tryparsamide therapy, of syphilis and ocular effect, (348).
and vitamin deficiency, (219).
Tubercle obstructing central retinal vein, (345).
Tuberculin, allergy, effect of, on trachomatous process, (1454).
consensual ocular desensitization to, (1450).
reaction in ophthalmology, 1067.
therapeutic, for ocular conditions, 86, (1213).
Tuberculoma of eye, (1472).
Tuberculosis, and eye disease, (345).
ocular, (1213), 1316.
allergy in, 1316.
of choroid, vitreous changes in, (976).
of conjunctiva, (89), (1454).
Parinaud's syndrome and, (1455).
experimental, (845), (1099), 1316.
human and, (1342).
iridocyclitis, (970).
metastatic, (1342), (1447).
panophthalmitis from, (95), (1331).
retinal periphlebitis, (830).
therapy of, antituberculin of Ymikov, (958).
irradiation, 1317.
rubrophen dye, (220), (1214).
toxic syndrome in, **1392.**
Van Lint test in metastatic, (1447).
vitamins A and C deficiency in, (835).
Tumors, (108), (353), (478), (600), (828), (1094), (1333).
of brain, see Brain.
of caruncle, (109), (1333).
of choroid, 569, (1095), (1337).
Tumors, (*continued*).
of choroid, (*continued*).
carcinoma, metastatic, mammary, (353), (478), (1335).
rectum, (840).
melanocarcinoma, (1096).
melanoma, malignant, 807, (1334).
with drusen formation of papilla, (346).
perithelioma, (354).
sarcoma, (840), (841).
of ciliary body, dictyoma, early, (1095).
melanoma, benign, (109).
myoma, (110).
of conjunctiva, Boeck's sarcoid, (225).
epithelioma, (1337).
pigmented, (1334).
hyperkeratosis, (591).
lymphoma, (109).
melanosis, malignant change in, 808.
papillomata, (108).
reticuloma, (1336).
sub-, lymphoid, (479).
of cornea, keloid, (601).
corneoconjunctival dermoid, 698.
cysts, see Cysts.
of eye, (601).
lymphocytoma, (354).
melanosarcoma, (838).
melanosis, (354), (838).
sarcoma in region of, (355).
tuberculoma, (1472).
of iris, Boeck's sarcoid, (98).
carcinoma, metastatic, 81, (355).
melanosis, (98).
of lacrimal gland, adenocarcinoma, (600).
Boeck's sarcoidosis of, (108), **675.**
of lacrimal sac, carcinoma, (479).
of lids, carcinoma, 74.
epithelioma, (354).
neurofibromatosis, (479).
neuromyoarterial glomus, (839).
of limbus, epithelioma, (1334).
of meibomian gland, carcinoma, (1096).
epithelioma, (351).
nasopharyngeal, ocular symptoms from, (833).
of oculomotor nerve, neurofibroma, (1334).
of optic nerve, 570, **1144.**
disc, angioendothelioma, (110).
phacoma, (1335).
extension of uveal melanoma into, (111).
glioma, 570.
meningioma, 570.
of orbit, **386, 497,** 1071, (1335), (1336).
angioma, (1335).
carcinoma, (355).
metastatic, mammary, (1094).
from prostate, (353).
chloroma, (1096).
fibrohemangioma, 75, (355).
fibroma, (110).
lymphoblastoma, follicular, (1334).
lymphoma, (600).
melanoma, malignant, (109).
neurinoma, (1337).
psammoma, **664,** (1095).

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts

Tumors, (*continued*).
of orbit, (*continued*).
removal, by transcranial attack, (839), 1071.
of plica semilunaris, nevocarcinoma, (225).
of retina, angiomatosis, (240), (841), (1095), (1336).
carcinoma, bilateral, metastatic, mammary, **63.**
diktyoma, (1095), (1336).
epithelioma, (840).
glioma, **1354.**
heredity in, (478).
prognosis, (355).
pseudo-, **164.**
treatment, diathermy coagulation, (839).
radium, (838).
melanosarcoma, (1094).
phakoma, (478).
of uvea, melanoma, malignant, (111), (840).
sarcoma, (1334).
prognosis, (355), 1064, (1096).
Twins, convergent strabismus in, (462).
ophthalmologic conditions in. 816.
refractive errors of, (1219).
Tyloma of tarsal conjunctiva, (591).
Tyndallimetry, limits of, in anterior chamber, **51,** 699.
Typhoid-H antibody concentration and experimental corneal ulcer, (92).

Ulcers, see Cornea.
Ultrashort-wave therapy in ophthalmology, (217), (1212).
Ultraviolet-light radiation for disease of lid border and cornea, (722).
United States, fifty years of ophthalmology in, (1101).
Naval Academy, myopia at, (1102).
Uvea, disease of, 1063.
ectropion, (232).
Uveal tract, sympathetic disease, and aqueous humor, (95), (231), (469), (595), (724), (969), (1080), (1226), (1461).
Uveitis, and alopecia, poliosis, etc., (233).
anoxia in, (470).
in arthritis, 701.
bilateral, with retinal detachment, (98).
glaucoma with, 702.
treatment, short wave, (1212).
vascular basis of, (470).
Uveoparotid fever, (1080).
and Boeck's sarcoid, (231).

Vaccine, gonococcal, (99).
Vaccinia, ocular involvement from, (94), (108), **257,** (590).
Van Millingen grafting, see Trichiasis.
Vasoneurosis with eye symptoms, (92).
Ventriculography, cortical blindness following, (833).
Verhoeff membrane, (1103).
Vestibulo-retinal reflex, (1229).
Vision, basis of, 1446.
color, blue-green region of the spectrum, (222).
defective, see Color blindness.
Vision, (*continued*).
color, (*continued*).
effect of, alcohol on, (599).
adrenalin on, (222).
influence of, in study of microscopic anatomy, (221).
in insects, (847).
tests, with polychromatic plates, (960).
and see Color plates.
and see Color blindness; and see Perception, and see Abstracts, section three.
defective, optical illusions from, (222).
effect of other sensory activities on, (220).
loss of, sudden, (957).
macular, sparing of, in occipital lobectomy, (475), (833).
night, testing, (1450).
and see Perception; and see Abstracts, section three.
Visual acuity, extrafoveal, as measured with Snellen test letters, **303.**
modification of, by protective colored lenses, (716).
requirements for Army, Navy, and aviation, 1438.
test of, for automobile drivers, (846).
Betts method, (115).
for large groups, (221), **779.**
charts, (1450).
colored, (461).
distance, for children, (1451).
illuminator for, **1061.**
in schools, (115), (606), **1170,** (1344).
Snellen, extrafoveal vision acuity measured by, **303.**
illumination standardization of, **287.**
measure of effect of reduced contrast on, (1217).
for industry, (1102).
near, chromatic, (718).
Visual cortex, angiospasm of, (106).
Visual fields, color, effect of sound on limits of, (960).
in experimental night blindness, (1451).
defects, brain tumors and, (834).
Visual localizations, neural mechanisms of, **1255.**
Visual mechanisms, critical intensities for visual flicker, (1450).
Visual threshold for blue, in youth, age, and aphakia, (716).
Visual tracts and centers, (105), (348), (475), (599), (832), (1091), (1471).
Visual tracts, 1207.
lesions of, third visual neuron, (1471).
Visual zone, 579.
Vitamins—
A, of blood, in adult gonorrheal conjunctivitis, (91).
and biophotometer test, (1211).
dark adaptation and, (826).
as measurement of status, (714).
B_2, (605), (1215).
C, cataract and, (238).
deficit—
A, (242), (604).
corneal necrosis and, (966).

Explanation: Numbers in heavy type refer to original articles, parenthetic numbers to abstracts.

Vitamins, (*continued*).
deficit, (*continued*):
A, (*continued*).
effect of, on dark adaptation, (344), 703, (1086).
hemeralopia and color fields in, (1451).
and keratoconus, 214.
ocular tuberculosis and, (835).
and visual acuity, (243).
B-complex, ocular conditions associated with, (481), (605), (1100), **1233, 1265.**
in corneal ulcer, (91).
C, ocular tuberculosis and, (835).
E, keratoconjunctivitis, etc., and, (1100).
effect of, on eye, (1342).
on light sense, (1219).
in lens, 69.
therapy, 1325.
A, (94).
burbot-liver oil intramuscularly, (715).
ointment, for epithelization, (219).
B, (1215).
B-complex, (1215), (1223).
B_1, tryparsamide reaction and, (219).
for corneal ulcer, (91).
K, in sprue-cataract postoperative hemorrhage, (1449).
C, for corneal inflammation, (1078).
for incipient senile cataract, (100).
P. P. (nicotinic acid), for vernal conjunctivitis, (1223).
Vitreous, adhesions of, with retina in retinal detachment, (824).
changes, in tuberculous choroiditis, (976).
cholesterin crystals in, (233).
detachment of, (1329).
experimental, **1349.**
globular, posterior, 1314.
traumatic, see Injuries.
expansion tendency curve of, (829).
hemorrhage, recurrent, (240).
zonule, and ciliary processes, (827).
and see Abstracts, section ten.
Vogt's localization of foreign bodies, (355).
Von Recklinghausen's disease, see Neurofibromatosis.

War, ophthalmic literature and, 711.
ophthalmic questions in, (115), (1102).
and see Injuries.
Washington University School of Medicine, retinal-detachment surgery in, results, 445.
Water-drinking test, see Glaucoma.
Weil's disease, ocular involvement in, (98).
Weil-Felix seroreaction in trachoma, (721).
White, Joseph A., 1208.
Wilson's hepato-lenticular degeneration, 559.
Wolfe, Claude Thomas, 955.
Wollaston and hemianopsia, **1053.**
Woodruff, Thomas Adams, 1209.
Wootton, Herbert Wright, 1072.

Xanthomatosis bulbi, (1092).
Xeroderma pigmentosum, ocular complications in, 570.
Xerophthalmia from biliary deficiency, (1225).
Xerosis in glass blowers, (593).
X-ray, examination, for diagnosis of intraocular foreign body, bloody, bone free, (1340).
in firearm injury of orbit, (1338).
injury, see Injury; and see Cataract.
therapy, for corneal ulcer, (91).
for iridocyclitis, tuberculous, (970).
for ocular tumor, lymphoma, (600).
for phlegmon of lacrimal sac, (1333).
for retinal-vessel disease, (241).

Ydmurt, ocular disease, etc., in, (1345).
Yearbook of the eye, ear, nose, and throat, 1940, 212.
Yellow oxide of mercury, bacterial properties of, (1214).

Zinc sulphate, bacterial properties of, (1214).
Zona, ophthalmic, see Herpes.
Zoster, see Herpes zoster.

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